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THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—11TH YEAR. SYDNEY: SATURDAY, MARCH 15, 1924. No .11.

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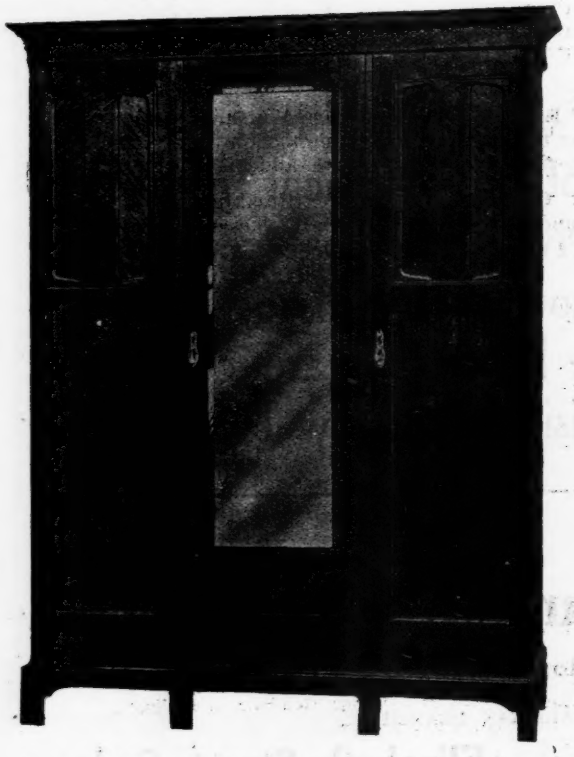

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THE MEDICAL JOURNAL OF AUSTRALIA

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A CASE OF ACUTE CARDIAC FAILURE SHOWING FLUTTER AND INCOMPLETE RIGHT BUNDLE BRANCH BLOCK WITH RECOVERY.

By W. N. HORSFALL, M.B., B.S. (Melbourne),
Sydney.

MR. A.B., aged fifty-three years, married, has lived on his means and has done no work for twenty-eight years; he has occupied his time in drinking beer.

He was first seen on April 9, 1923, in hospital, where he had been admitted on February 2, 1923, with cardiac failure and dropsy. He complained of shortness of breath. On April 9 he was suffering from heart failure with œdema of the feet and legs and thighs, with much fluid in the abdomen and œdema over the chest and arms. The skin pitted on pressure two finger breadths below the angles of the scapulæ. He was completely water-logged.

He could not rest lying down and suffered from attacks of increased dyspnœa on awakening at night.

His pulse could not be felt at the wrist. The cardiac impulse could not be felt on palpation. The heart sounds were very distant and no murmur could be heard. The heart rate, counted during auscultation, was one hundred and twenty per minute and it was regular.

Previous History.

Before 1917 he was a healthy man. In that year he had an illness lasting three months and suffered from cough and bronchitis. In 1918 he was in hospital for six weeks with Bright's disease. He states that dropsy was present then.

Since 1918 he has been comparatively well, until the beginning of 1923, when acute heart failure set in with congestion. He has no history of rheumatism and has not suffered from angina.

On April 10 an electro-cardiogram was taken and a condition of a 2:1 auricular flutter was discovered (Figure I.). The curve shows a regular heart rate of one hundred and twenty-five per minute. In addition there was present an incomplete block of the right branch of the auriculo-ventricular bundle.

He was given tincture of digitalis with the hope that the flutter would turn into fibrillation and then revert to a normal rhythm or if the normal rhythm did not eventuate, that we could control his ventricular rate in fibrillation with digitalis. The tincture of digitalis did not agree with him. His condition becoming worse after 3.5 mils (one drachm) had been given and the same thing happening after many attempts, the tincture was given up and Guy's pill (mercury, squills and digitalis) was substituted, one pill three times daily. He began to improve and with the improvement I thought it may not have been the digitalis which was the cause, but the mercury in the Guy's pill and that we had to deal with a case of a syphilitic lesion in the heart.

Later History.

On October 9, 1923, I obtained permission to see him again in his home (in Surry Hills, Sydney). He was able to walk about in his home and showed no breathlessness when so doing. He slept well. His pulse rate was sixty per minute and irregular when counted at the wrist. Extra-systoles accounted for the irregularity.

The apex beat was 2.5 centimetres (one inch) outside the nipple line and in the fifth intercostal space. The liver was felt one hand's breadth below the right costal margin.

The veins of the neck were not visible in a reclining posture. No crepitations were heard at the bases of the lungs. All œdema had disappeared. The pupils reacted to light. His systolic blood pressure was one hundred and sixty-eight millimetres of mercury. His condition was greatly changed and improved.

He had a large rupial ulcer over the antero-lateral surface of the right leg in its lower third which was not present in April. The ulcer was six centimetres long and four centimetres wide, its length being in the long diameter of the leg. He showed well marked *lineæ albicantes* over the abdomen, the folds of the axillæ, on the inner side of both arms and on the upper part of the thighs.

Electro-cardiograms were obtained on October 13, 1923 (Figures III. and IV.). These showed a normal sino-auricular rhythm with extra-systoles, some arising in the right ventricle and others arising low down in the auriculo-ventricular nodal tissues. The flutter had gone, but he still showed incomplete right bundle block.

On October 24, 1923, a Wassermann test was carried out, but no reaction was obtained.

Discussion of the Clinical Case.

The patient has recovered. He says his heart is as good as ever it was, but we know that it is not. Why did he recover? At the beginning we had a patient who exhibited the signs of auricular flutter with signs and symptoms of acute cardiac failure, now the patient shows an absence of flutter and an absence of the signs and symptoms of acute cardiac failure.

It is probable that the digitalis in the Guy's pill had turned his flutter into fibrillation and that then the heart had reverted to a normal rhythm. But what is the reason that the tincture of digitalis did not bring about this result? I do not know. It is possible that the quality of the tincture of digitalis may have been at fault. Digitalis deteriorates after a certain length of time. It becomes inert and loses its potency. Are we to look on the relief of this man's condition as due to the beneficial results of an alteration of the rhythm of flutter to a normal sino-auricular rhythm? I think we must. At the same time I cannot resist the inclination to regard his condition primarily as cardiac syphilis.

The conduction in the ventricle shows no material change (compare Figures III. and IV. with Figure I.), The presence of a rupial ulcer on the leg would of itself suggest a syphilitic lesion. For a long time I have considered such ulcers to be tertiary syphilides. That there was no response to the Wassermann test should not influence us unduly, as he has been taking mercury in doses of 0.06 gramme (one grain) three times a day from April to October in the Guy's pill. The digitalis in the Guy's pill, by altering the rhythm, must be held responsible for the improved condition. The mercury may have aided this action in a specific direction. The Guy's pill is an old pill. It has been used for many years empirically and now at great pains we are trying to demonstrate why it had such a reputation among the old school. There is no proof that the action of mercury is specific in this case. The question of syphilis must remain only an opinion.

The case proves the value of an electro-cardiographic examination. Without such an examination the condition of flutter would have passed unnoticed. It was impossible to determine by a bed-side examination.

Discussion of the Electro-Cardiographic Curves.

Auricular Flutter.

The recognition of auricular flutter clinically is at times very difficult, especially in those cases with a not unusually fast heart rate. At ventricular rates of one hundred and forty or one hundred and sixty one is naturally expectant of a changed rhythm such as flutter. If the flutter is one of 4 : 1 rhythm with a regular ventricular rate of seventy, the difficulty of discovering the condition by clinical means is even greater.

Even with the aid of an electro-cardiograph the recognition of a 2 : 1 flutter often is difficult, for the reason that one *P* wave is lost in the previous ventricular complex. It is characteristic of flutter that the heart rate observes a definite regularity from cycle to cycle, there being less than one hundredth of a second's difference from one ventricular systole to the next in a case of 2 : 1 flutter.[1]

In Figure I. we can recognize one *P* wave clearly in lead ii. It is indicated as P^1 and its apex is slightly more than 0.1 second before the beginning of *R*. On studying the portion of the curve indicated at P^2, it was thought that another *P* wave was present. Suspicion grew stronger when the distances from $P^2 - P^1$ and from $P^1 - P^3$ and again from $P^3 - P^4$ were measured. These distances are regularly spaced. Another curve was taken by means of chest leads (Figure II.). Copper discs, five centimetres in diameter, were affixed to the chest by means of strapping. The discs were coated with a layer of flour and salt water made into a paste. One lead was attached to the front of the chest wall close to the sternum in the second left intercostal space, the other lead was attached five centimetres below the left nipple and two and a half centimetres outside the left nipple line. Figure II. shows the record obtained and the more definite auricular waves. I have indicated in two of the cycles the course which would have been taken by the auricular oscillations if they had not been interfered with by the ventricular complex. The case is one of flutter with auricular oscillations of two hundred and fifty per minute. A continuous circular wave is proceeding in some part of the auricle and only every alternate impulse reaches the ventricle.

Incomplete Right Bundle Branch Block.

It is characteristic of a complete lesion of the right division of the bundle that the *Q.R.S.* complex is prolonged, often notched and its amplitude is increased. *T* is exaggerated in amplitude and is opposite in direction to the main initial deflection of the ventricular complex that is *Q.R.S.* With an upright *R* we associate a downward directed *T*. With *S* predominant we associate an upright *T* wave.[2]

This knowledge has been obtained by experimentally dividing the right or left division of the bundle.

Figure V. is from a patient with defective conduction along the right division of the bundle[2] and closely resembles the curves obtained experimentally.

Figure I. which is a record of our patient, shows some resemblances, but there are important differences.

The *Q.R.S.* complex averages normally 0.08 second and should not exceed 0.1 second. In Figure I. it is just over 0.1 second. The *Q.R.S.* complex is notched, but does not show the amplitude of Figure V. In Figure I. *T* conforms in direction to the curves as produced experimentally, but does not show an increased amplitude. In lead ii. of Figure I., *T* is in the position where indicated and must not be confused with the depression immediately following it. This is proved by measuring from the beginning of *R* to *T* in the other leads and measuring the same distance from *R* in lead ii.

Now curves like Figure I. have been described previously and have been called right bundle block. We are not safe in making such an assumption, unless we produce a curve clinically which conforms in every respect to that obtained by experiment. With the results of experiment as our guide we are on safe ground.

Many writers have tried to associate certain abnormal curves with certain cardiac lesions. They have attempted to diagnose a definite pathological state from an abnormal curve. We can, of course, prove a disturbance of function, but so far there is not much evidence advanced to prove the association of disturbed function with a definite pathological lesion.

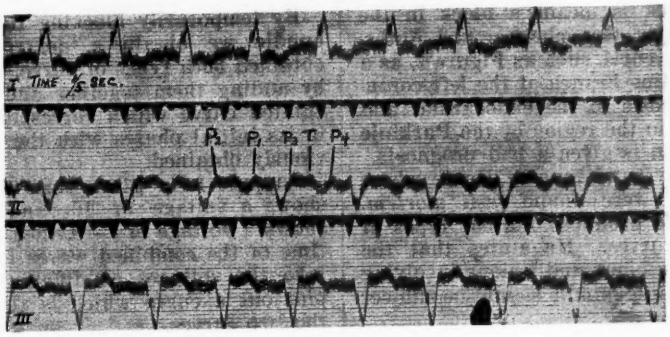

FIGURE I.

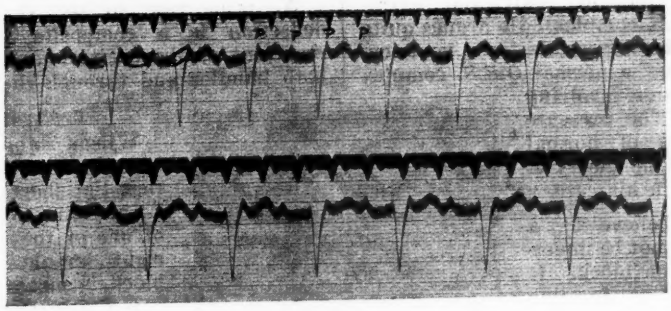

FIGURE II.

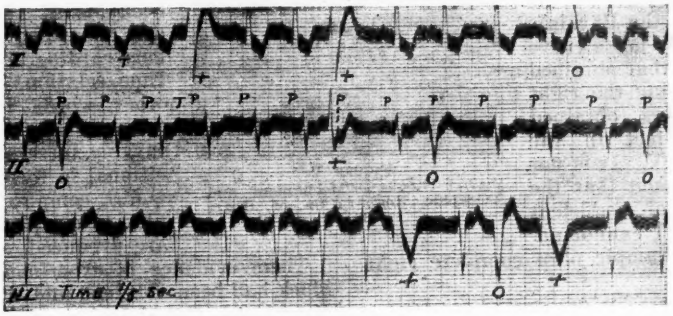

FIGURE III.

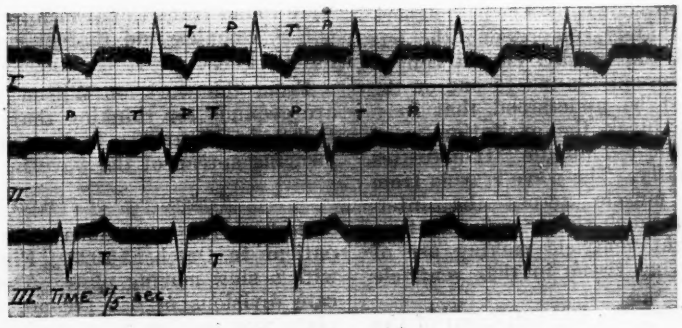

FIGURE IV.

Oppenheimer and Rothschild[3] have described such curves as Figure I., as cases of arborization block. It must be remembered that the impulse causing the ventricles to contract passes from the auriculoventricular node down the auriculoventricular bundle in a normal subject. The bundle divides into two main branches, one of which supplies each ventricle. Each ventricle becomes activated by the impulse spreading with great speed over the whole interior of the ventricles. It spreads through the Purkinje network or arborization, so that the ventricular muscle contracts almost simultaneously and the spread of the wave in the ventricular muscle is from within outwards. The speed of travel in the Purkinje network is ten times that in the ventricular muscle.

By arborization block is meant a block in the arborization of Purkinje. Oppenheimer and Rothschild would interpret a curve such as Figure I. as a sclerosis of the descending branch of the left coronary artery and a sclerosis in the endocardial and sub-endocardial layers in the region of the Purkinje network. The condition is given a bad prognosis.

Drury[4] on the other hand reports a case where lesions just described were found *post mortem*. The patient during life produced normal types of electro-cardiographic curves. He states that the question of arborization block and lesions involving the smaller branches of the bundle should be subject to revision.

Hewlett[5] reports a case and shows a curve whose ventricular complexes are analogous to Figure I. His patient did not exhibit flutter. In addition there were frequent ventricular extra-systoles of normal *Q.R.S.* complex. He argues quite rightly that it would be impossible for a ventricular extra-systole to show a normal *Q.R.S.* complex in the presence of a lesion involving the Purkinje tissue. The presence of these extra-systoles of normal outline is thus against the theory of an arborization block in his case.

I am thus forced to believe that a curve like Figure I. cannot be understood as an arborization block. It cannot be understood as forming proof of the existence of a pathological lesion, such as sclerosis of the descending branch of the left coronary artery or sclerosis of the endocardium involving the Purkinje network. It is certainly not a curve strictly comparable to that obtained experimentally by cutting the right bundle[2] or to curves obtained clinically showing defect in the right bundle as Figure V. It is not comparable for the reason that the deflections are not of sufficient amplitude. Therefore, I cannot admit Figure I. as an example of a complete right bundle block.

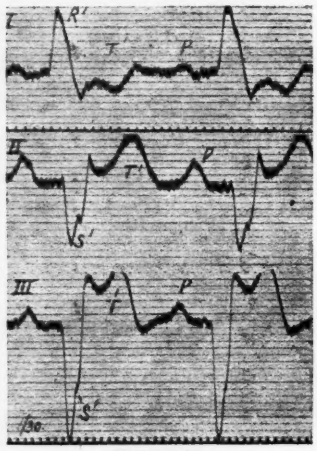

FIGURE V.
After Lewis: "Mechanism and Graphic Registration of Heart Beat."

The Normal Q.R.S. Complex.

The normal *Q.R.S.* complex represents the spread of the excitation wave down both divisions of the bundle and by Purkinje tissue through the ventricular muscle. It has been shown by Lewis[2] experimentally that by alternately compressing the left and right divisions of the bundle, curves can be obtained showing the spread limited to one or the other bundle. It must be understood that in a complete right bundle block the excitation wave can only travel down the left bundle. It activates the left ventricle normally. The wave travels from the left ventricular muscle across the inter-ventricular septum at a much slower rate and then activates later the right ventricle making use of the serviceable conducting tissue in that ventricle below the block.

By temporarily blocking the right division of the bundle we obtain what is called a lævo-cardiogram.

By temporarily blocking the left division of the bundle we obtain a dextro-cardiogram. Having obtained both types of curve, we can combine them by adding them together and thus produce a calculated curve which corresponds fairly accurately in its initial phases with the normal curve as previously obtained.

Suppose at a given time the dextro-cardiogram shows a voltage of 10, and the lævo-cardiogram shows a voltage of — 8, then the resultant at that time of the combined waves is + 2. We can then take the voltages at different time intervals and plot out a curve which is the algebraical sum of the two curves.

Figure VI. will demonstrate the matter clearly. The curve as calculated approximates to the normal curve as obtained by experiment.[2]

We come now to see that the normal *Q.R.S.* deflection is a composite picture showing the resultant of the excitation waves travelling down both bundles and through the ventricles.

Explanation of Figure I.

Wilson and Hermann[6] have brought forward evidence to show that the aberrant curves, as Figure I., are due to an incomplete block of one of the main divisions of the right bundle. By an incomplete block is meant delayed conduction.

If the dextro-cardiogram be added to the lævo-cardiogram in their proper time relations a normal *Q.R.S.* complex is produced as has been stated (Figure VI.). If the dextro-cardiogram be added to the lævo-cardiogram not in its proper time relation, but a little later, in other words if the dextro-cardiogram is shifted 0.005 second or varying intervals later, curves transitional in form between the normal *Q.R.S.* complex and the *Q.R.S.* complex of complete right bundle block are produced, as in Figure V.

Their evidence is experimental. Right bundle block had been obtained and the right ventricle was stimulated rhythmically at rates a little slower than the heart rate. The super-imposed effects of dextro- and lævo-cardiograms were thus obtained at varying time intervals and *Q.R.S.* complexes were found transitional in form between the normal *Q.R.S.* complex and that of complete right or left bundle block.

From the work of Wilson and Hermann there are grounds for thinking that the notching of the *Q.R.S.* in Figure I. is associated with the commencing spread of the wave in the right ventricle.

Explanations of Figures III. and IV.

Both curves were taken after the recovery of my patient in October, 1923. Figure III. was taken with a slow moving plate. The vertical lines indicate divisions of 0.2 second. In Figure IV. the plate was moving more rapidly in order to make the curves comparable to Figure I.

The same delayed conduction down the right bundle in Figures III. and IV. is seen, as was seen in Figure I. The ventricular rate is about 80 per minute. The *Q.R.S.* complex is wider than normal. For this reason it is not correct to say that the curves are examples of left ventricular preponderance. Also *T* is distinctly downward in lead i. and is opposite in sign to the main initial deflection.

There are extra-systoles arising in the right ventricle and indicated as + in Figure III.

The complexes indicated as *O* in Figure III. are extra-systoles. They follow the preceding complexes at a shorter interval. Now it is the rule that an extra-systole arising in a supra-ventricular focus causes no alteration in the *Q.R.S.* complex. An extra-systole arising in any part of the ventricle naturally deforms the normal *Q.R.S.* complex. These extra-systoles indicated as *O* do cause an alteration in the *Q.R.S.* complex. At first sight they may be thought to be ventricular extra-systoles. Nevertheless, I think we can say that they arise from a supra-ventricular focus. They arise low down in the auriculo-ventricular node. These cycles *O* follow the preceding cycle at a shorter interval. Following at a shorter interval, the conduction in the right bundle, being already delayed, is delayed still further and the *Q.R.S.* complex is widened still further. Further, if the ventricular complexes of the fast moving plate of Figure IV. are compared, it will be seen that there are minor differences in amplitude from one to another noticeable in leads i. and iii. It is considered that these minor differences in amplitude are due to minor differences in conduction down the right bundle. The presence of a nodal extra-systole accentuates this difference to a greater degree.

We are able to pick out *P* waves in such complexes indicated as *O* in Figure III. The *P* waves space evenly. The ventricular wave on these occasions originated not in the sino-auricular node, but low down in the auriculo-ventricular node. They are nodal extra-systoles. The auricles contract in response to an impulse from the sino-auricular node, but before this impulse can reach the ventricles, the ventricles have already become activated from an impulse which originated in the auriculo-ventricular node. Both auricles and ventricles are contracting at the same time. If the ventricular impulse had activated the auricles by a reverse conduction through the auricular-ventricular node, the *P* waves would not space evenly and the *P* waves, especially the one buried in the complex + of lead ii. in Figure III., would not be upright.

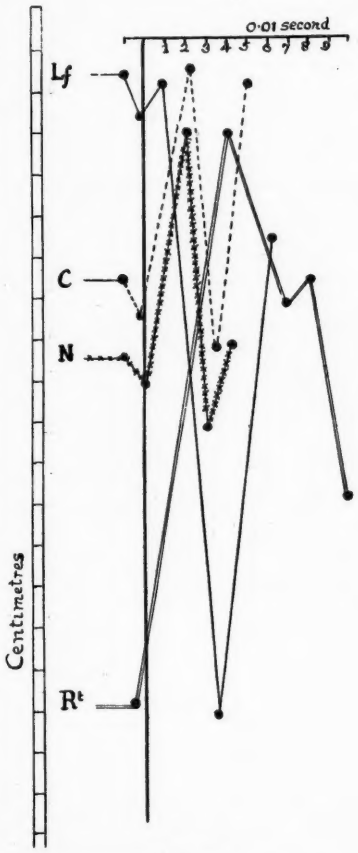

FIGURE VI.
Lf represents a curve obtained when the right division of the bundle is blocked—a lævo-cardiogram. *Rt* represents a curve obtained when the left division of the bundle is blocked —a dextro-cardiogram. *N* is the normal curve obtained from the experiment. *C* is the curve of the algebraical sum of *Lf* and *Rt*.

The *P.R.* interval of the regular cycles is 0.2 second. From the beginning of *R* to the beginning of *P* in the complex just mentioned, it is about 0.1 second. A reverse conduction could not go so quickly.

It cannot be reasoned that this particular extra-systole is nodal in origin and due to delayed conduction in the left bundle. It has the characteristics of a right ventricular extra-systole similar to those marked + in leads i. and iii.

We are justified, I think, in calling these complexes indicated as *O* in Figure III. nodal extra-systoles. They are not typical of ventricular extra-systoles. It is significant that in these complexes *T* is larger and the *S* wave wave is deeper. It is significant also that they are not exactly alike. These facts suggest a transition between the normal *Q.R.S.* complex of a normal curve and the *Q.R.S.* complex of a fully developed right bundle block of Figure V. The transition of one to the other is due to a gradual shifting later of the dextro-cardiogram. In other words, it is fair to assume that a variability in the degree of block in the right bundle is the cause of these *Q.R.S.* deflections being wider in those indicated as *O*. They are wider than the other cycles of this curve. These in their turn are wider than normal curves.

It is thus found that there is clinical evidence in support of the view of Wilson and Hermann[6] previously mentioned, that curves like Figure I. are due to a shift in the relative positions of the dextro-cardiograms and the lævo-cardiogram, that this shift depends on delayed conduction down the right bundle. The evidence in support of their view rests on the fact that when the conduction of the right bundle is already strained, if it is strained still further by the passage of another impulse at a shorter interval due to the occurrence of a nodal extra-systole, we then obtain a changed ventricular complex. It is changed by the further delay in the dextro-cardiogram relative to the lævo-cardiogram and the changed complex more nearly approaches that obtained in complete block of the right bundle.

A curve was taken from chest leads in October, 1923, similarly to that taken in April, 1923, as Figure II. The curve is not reproduced. It does not show auricular flutter to be present.

Conclusion.

This case is recognized as an incomplete right bundle block, meaning by that a delayed conduction in the right bundle. We are not justified from the curve alone in saying anything further as to the cause of that block.

It is wrong in my opinion to make a diagnosis of a pathological lesion and further to arrive at a prognosis from an examination of such an electrocardiogram alone. How can one say whether a widespread sclerosis, a large gumma or a comparatively slight lesion has involved the right bundle?

If we have an ordinary case of partial auriculoventricular block, we do not necessarily think of gross lesions as the cause. We know very often they are not the cause.

Many workers have compiled statistics showing the mortality associated with various abnormalities found in the ventricular complexes, curves like Figure I. amongst them.

As we all know statistics may be very misleading and in this case I think they are, as they are based on a wrong premise. Although an unusual curve certainly indicates an unusual or faulty conduction, still, this may not be associated with gross cardiac lesions. Hence it seems to me that statistics of mortality based on errors of function which may not be associated with gross cardiac lesions, are inaccurate. Further, I think that basing mortality statistics on the reading of abnormal electrocardiograms is a misinterpretation of the uses of the electro-cardiograph and liable to bring the instrument into disrepute. The electro-cardiograph is a necessary instrument of investigation of some cardiac diseases, as this case clearly demonstrates. It has its limitations and those of us who use the instrument, should define and observe such limitations.

I am continually being asked the question: "What do you do with the electro-cardiograph?" This case and others answer that question. Then follows a second question: "When you have done all that, what more can you do for the patient?" Sometimes nothing; sometimes a great deal. The second question should never be put by one who considers himself a student of medicine.

The aim of practical medicine is to establish a complete diagnosis, to arrive at the ætiology of the disease and then we are better able to launch out to a correct prognosis and to apply appropriate treatment. To achieve this aim we have the use of our fingers and eyes which will always remain our greatest instruments. In addition we have the thermometer and stethoscope and the laboratory. The polygraph and electro-cardiograph of recent birth have their place also within certain limits.

Summary.

A case of extreme heart failure with congestion is described. Electro-cardiograms show auricular flutter and an incomplete right bundle block. Reasons are advanced in support of this interpretation. The patient recovered and reverted to a normal cardiac rhythm. The condition of incomplete right bundle block persisted.

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GASTRO-ENTEROSTOMY: OBSERVATIONS ON ITS MECHANISM AND ON THE PRODUCTION OF PAIN IN DUODENAL ULCER.

By FRANK L. APPERLY, M.A., M.D. (Oxon.),
Acting Professor of Pathology,
University of Melbourne.

WITH the object of throwing further light on the mechanism of the gastro-intestinal tract in disease and incidentally on the mechanism of gastro-enterostomy itself, twenty-seven patients on whom this operation has been performed, have been examined by the fractional test-meal during the last eighteen months at the Melbourne Hospital. Thirteen of these were also examined before operation. The results show a very striking division into two clearly defined groups: (*a*) those in whom the free acidity is reduced completely or almost to nil and (*b*) those in whom the acidity curve remains as high or even higher than before operation. These results are set out in Table I. and Table II. (see pages 260 and 261).

Group (*a*).—In this group which includes sixteen patients, alkali has entered the stomach in sufficient amount to neutralize all or nearly all free acid, in spite of the fact that absence of free acid might mean a diminished rate of output of pancreatic alkali owing to diminished activation of duodenal prosecretin. When peristalsis opens both pylorus and stoma (the latter by contraction of the circular muscle fibres cut across in making the stoma) acid chyme is projected into the duodenum and jejunum. The rapid rise of tension in these organs stimulates contraction, with return of portion of the food *plus* duodenal alkali to the stomach and consequent neutralization of its contents. When the jejunal muscle is highly irritable, the return of its contents to the stomach may be so rapid as to lead to vomiting. Direct experiment has shown that rapid distension of the jejunum alone produces vomiting. This may account for some of those cases of vomiting occurring after operation.

Group (*b*).—In this group, comprised of eleven patients, the acidity of the gastric contents is not appreciably diminished; it remains high and in some patients is even higher than before operation. Alkali has, therefore, failed to enter the stomach in sufficient amount to produce complete neutralization as it normally would do. Why is this?

Failure of neutralization in these cases may be due to: (i.) Hypersecretion of acid; (ii.) defective production of pancreatic alkali; (iii.) inability of sufficient alkali to enter the stomach owing to (*a*) obstruction at the stoma, (*b*) hypertonus of the stomach, (*c*) defective jejunal reflux forces.

Hypersecretion of Acid.

Until comparatively recently it has always been assumed that variations in acidity depended on variations in secretion of gastric juice, but the work of Bolton and Goodhart[1] and others including the writer[2] has shown that these variations are really due to varying degrees of neutralization and dilution of gastric contents brought about by the regurgitation of alkaline duodenal juices. Although I know of no proof that hypersecretion ever does occur, it is nevertheless probable that there are variations in secretion in healthy gastric mucous membranes. Should such a hypersecretion take place, however, there would inevitably be a corresponding hypersecretion of pancreatic alkali, since free acid in the duodenum will always stimulate the pancreas provided the latter is healthy. Sooner or later this alkali is regurgitated into the stomach, this phenomenon depending on the irritability of the jejunum. It follows then that, if complete neutralization of gastric contents does not take place, the fault lies in the jejunal reflux forces and not in the amount of gastric secretion, provided always that the passage between stomach and jejunum is clear and that the stomach is emptying.

The question, however, has been put to direct test by estimation of the total chlorides in each of the test-meal fractions of seven patients [three from Group (*a*) and four from Group (*b*)] and comparing the results. It has been stated that the strength of the total chlorides is a true measure of secretion, but when we consider that this strength must be considerably lowered by the dilution produced by excessive neutralization, it is clear that this is not true. Without going into details, it may be stated that the amount of this dilution can be calculated from the total chloride and free acid in each fraction and then by making the necessary correction to the total chloride estimation we can arrive at a true comparative measure of secretion. Although there was some overlapping in the results, when the averages of each group were plotted (see Figure I.), calculation showed that though the chloride curve of Group (*a*) was lower than that of Group (*b*), the difference was entirely due to the greater dilution undergone by the gastric contents in Group (*a*). The secretion was, therefore, the same in both groups.

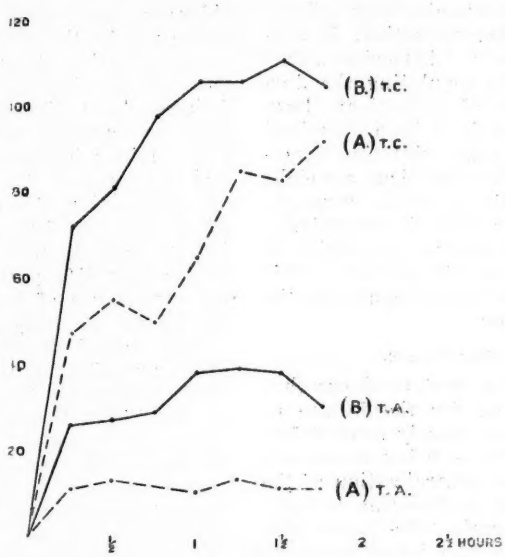

FIGURE I.
T.C. = Total Chloride. T.A. = Total Acid.

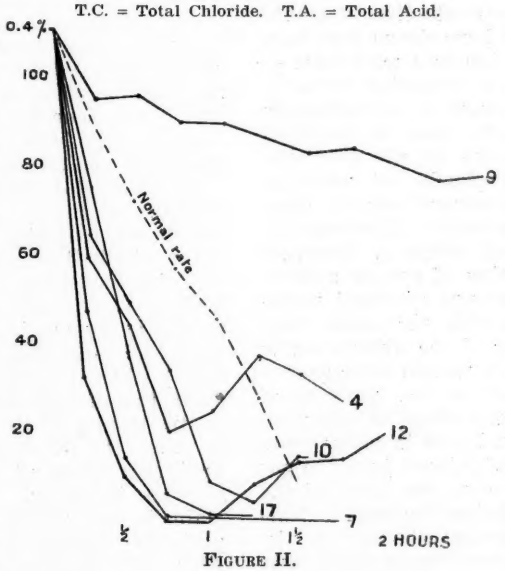

FIGURE II.

Defective Production of Pancreatic Alkali.

To find if there were some defect in the output of pancreatic alkali, a possibility since pancreatic disease and duodenal ulcer are sometimes associated, the rates of neutralization of a fixed volume of 0.4% hydrochloric acid placed in the stomachs of five of these patients [two from Group (*a*) and three from Group (*b*)] were determined, as described in a previous paper in this journal.[3] It was found that the hydrochloric acid was neutralized even more rapidly than in normal people, showing, therefore, that pancreatic alkali is certainly produced in abundance (see Figure II.). The figures are those of the case numbers.

Obstruction of Alkali at Stoma.

If there were some obstruction at the stoma in Group (*b*) preventing reflux or alkali, then there would also be some retardation in the rate of gastric evacuation. It was found, however, that the emptying times after gastro-enterostomy alone in each of the groups (*a*) and (*b*) average 1.4 hours, whereas if the patients subjected to Pólya's operation were included the average time in Group (*b*) was a little less than that of Group (*a*). Hence the possibility of an

obstructive influence can be eliminated. The effect of operation on the emptying time will be discussed later.

Hypertonus of the Stomach Preventing Reflux of Alkali.

Another possibility is that a high gastric tonus may exert such a pressure on the stomach contents that the jejunal forces will be unable to overcome this tonus, a small contracted stomach, for example, having no room for regurgitated duodenal fluids. That high tonicity often accompanies high acidity which is due to defective neutralization, is suggestive and in a previous paper[2] I have shown that tonus is probably a factor in regulating the duodenal reflux. An examination of my patients, however, shows that of those who have been examined by means of X-rays there is none exhibiting hypertonus in either group. In Group (*a*) there are eight orthotonic and four hypotonic or atonic stomachs and in Group (*b*) six and five of each respectively, that is, Group (*b*) contains a smaller proportion of good tone stomachs than does Group (*a*). This eliminates gastric tone as the factor regulating the duodenal reflux in these cases.

Defective Jejunal Reflux Forces.

The elimination of the above factors shows that there can be only one reason for the failure of alkalis to enter the duodenum, namely some defect in the reflux forces. That these reflux forces are normally present and produce neutralization of the gastric acid towards the end of digestion is daily demonstrated in test-meal work. We know also that when the irritability of the intestine is increased, as in enteritis, these reflux forces are greatly exaggerated with the production of vomiting. In a previous paper[4] I have shown that false or apparent achlorhydria is due to a mild form of the same phenomenon, that an excessive reflux of intestinal fluids occurs with complete neutralization of gastric acid in cases where there is increased intestinal irritability, as shown by the fact that intermittent diarrhœa and attacks of vomiting occurred in 55% and 50% respectively of these patients. I have also demonstrated[5] that retching alone produces an increased reflux of duodenal juices with a constant lowering of gastric acidity.

On the other hand, the amount of alkali regurgitated may be diminished with the result that hyperacidity is produced and if the diminution is marked, high acidity may even remain after gastro-enterostomy, as has occurred in the patients of Group (b). The fact that the shape of the post-operative acidity curve is unaltered in many cases shows that whatever reflux of jejunal juices might occur, it does not reach even to the level of the stoma and that the fault is below the stoma. This state of affairs might be brought about: (i.) by actual depression of the jejunal forces which produce the reflux, or (ii.) by an increased irritability of the duodenum or upper part of the jejunum in which case the returning fluids would re-initiate peristalsis, thus neutralizing the effect of the reflux forces lower down. In the former case we would expect a slower passage through the intestine, in the latter an accelerated passage.

The irritability of the duodenum and upper part of the jejunum can be raised, as can that of any part of the intestine, as Alvarez[11] has pointed out, by some local lesion. Overaction of the vagus, absolute or relative, reflex or central, appears to have the same effect in the upper portions of the intestine and indeed it is probable that this vagal action precedes duodenal ulcer. Hence we would expect a defective reflux of alkali into the stomach in duodenal ulcer before operation and, when considerable, even after gastro-enterostomy. This is borne out by the facts. Thus Guy[6] has found in an examination of twenty-six patients after gastro-enterostomy that operation for gastric ulcer was followed by a disappearance of the free acid, but that for duodenal ulcer resulted in an unchanged or even in a higher acidity than before. Hurst[7] and Conybeare[8] found the same and Hunter[9] in an examination of twenty-seven patients before and after operation found that the acidity fell to zero in 71% patients with gastric ulcer after operation, but in only 31% patients with duodenal ulcer. In my own series of twenty-one demonstrated ulcers the figures are respectively 80% and 45%.

That actual depression of the jejunal forces is also a probable factor is shown by the X-ray records of the patients in Group (*b*). In these we find that at six hours the head of the barium meal has reached the terminal ileum in three and the caecum in one of the four patients in whom this point is shown, whereas in Group (*a*) the head has gone much further, reaching points between the caecum and transverse colon in eight out of nine patients and the terminal ileum in only one. Yet at twenty-four and forty-eight hours there is no difference between the groups. If these patients can be taken as typical, then it would appear that the barium movement is retarded along the jejuno-ileum, that the fault lies there and is due either to poor jejunal tone or to poor response to the stimulus of acid chyme or to both. We, therefore, have another reason for the failure of the neutralizing forces.

The above deductions now give us an explanation of many of the symptoms in duodenal ulcer and certain other dyspeptic conditions. A defect in the reflux of alkali not only into the stomach but even into the duodenum and upper part of the jejunum leads to (i.) defective neutralization of gastric acid producing hyperacidity, particularly at the end of digestion (and this is very typical of duodenal ulcer) when the stimulus to the jejunum of larger quantities of acid food is wanting; (ii.) defective neutralization of the last portions of gastric acid to enter the duodenal bulb, which remains there producing spasm of the pylorus and pain when the stomach is empty. Hurst[10] states that acid remains in the bulb when the stomach is empty, which probably depends on the fact demonstrated by Alvarez[11] that the irritability of the duodenal bulb is very low. It is generally conceded that pain in duodenal ulcer is due to pyloric spasm. It also leads to (iii.) a defective neutralization of gastric acid even after gastro-enterostomy with little or no change in the acidity graph.

The above also explains (iv.) the hunger pain in certain reflex dyspepsias in which the irritability of

the duodenum and upper part of the jejunum is raised reflexly *viâ* the vagus nerve, so that the reflux forces are blocked; (v.) the occasional hunger pain associated with pancreatic defect where there is a diminished production of alkali for neutralization of acid in the duodenum and (vi.) the part played by gastro-enterostomy (see below).

The Rôle of Gastro-Enterostomy.

We are now in a position to appreciate the part played by a gastro-enterostomy in the relief of pain and the neutralization of duodenal acid in duodenal ulcer. High gastric acidity *per se* has little or nothing to do with the production of pain, as can readily be shown by introducing acid into the stomach. Normal people and the patients who provided the curves shown in Figure II. were quite comfortable with 0.4% hydrochloric acid in their stomachs. This high acid if anything would tend to relax the pylorus and thus relieve any pain present. On the other hand acid fluid in the duodenum, as in duodenal ulcer, produces closure of the pylorus and if it be highly acid and not neutralized rapidly, especially when the pyloric reflex arc is in an irritable condition, spasm and pain. Incidentally it is interesting to note that when acid was introduced into the stomach of Case 9 (pancreatitis with fat necrosis) just before operation, pain appeared and persisted (see Figure II.).

We can attack the cause of this pain in several ways. The usual method is to give alkalis to neutralize or otherwise bind the gastric acid towards the end of digestion. This neutralized fluid then is the material which occupies the duodenal cap when the stomach is empty. Or we may give drugs which are stimulating to the jejunum so that the reflux forces are augmented and alkali is driven upwards with neutralization of duodenal acid. Olive oil has this effect and there is evidence that the emetic drugs in small doses may have the same effect.[12]

The best plan of all, however, would be to utilize where suitable some natural driving force of the body to augment these defective jejunal forces and I now look upon gastro-enterostomy as such a device. By making the stomach open into the intestine below the point of entry of the pancreatic juice, gastric contents projected by the expulsive forces of the stomach into the intestine below this point are driven partly up the duodenum carrying alkali before it into the upper part of the duodenum, with consequent neutralization of acid lying there, and relief of pyloric spasm and pain. For success then the stomach must possess sufficient driving power to accomplish this. This is probably one of the most important reasons for the persistence of symptoms in patients with duodenal ulcer in whom the stomach possesses poor peristaltic power, or is atonic or hypotonic or there is obstruction at the stoma.

Perhaps medical treatment might have more success if we used more of those drugs such as olive oil which stimulate jejunal activity. In this connexion the work of Alvarez[12] on the action of drugs on the intestine may be of very great value in the near future. My own experience along these lines, although distinctly encouraging, is so far too short to be worth recording.

Other Causes of High Acidity After Operation.

Turning now to those patients in whom obstruction of the stoma is present, we will also find high acidity after operation, for reasons already discussed. These are the patients presenting symptoms after operation, due, of course, to interference with the augmenting power of the stomach. The position then is very much the same as it was before operation. Such an obstruction of the stoma may be absolute or partial owing to (i.) organic causes, such as cicatricial stenoses, or (ii.) spasm, such as by gastro-jejunal or jejunal ulcer or undue irritability of the jejunal wall.

In my own series there is no case representing cicatricial stenosis of the stoma, but Case 8 might be explained by spasm at the stoma if such a condition does occur, as asserted by Spriggs.[13] Thus a month after operation, X-ray examination showed the stomach half-full at six hours, whereas judged by the fractional test-meal, emptying was certainly very rapid, as shown by the fact that at three-quarters of an hour strongly bile-stained fluid containing no food was withdrawn. Presumably the barium which is said to have a stimulant action on the stomach, had initiated a spasm at the stoma, whereas the gruel of the test-meal which is very bland and non-irritating, appears to have passed through the stoma without producing this spasm or only an intermittent one. There was also a high acidity with a series of steep falls indicating an intermittent inrush of large gushes of alkali and suggesting intermittent spasm. A second test-meal examination six weeks later showed almost complete neutralization for an hour, followed by a sudden high rise, presumably another spasm. This explanation of the great difference of emptying times exhibited in some cases by barium and test-meal gruel might be applied to the condition of several other patients, a point which might become useful in the diagnosis of jejunal ulcer. As spasm of gastric circular muscle fibres would have only the effect of opening the stoma still wider, it is more probable that the stoma becomes closed by the simultaneous contraction of both the gastric and intestinal circular muscle fibres which have been cut across in making the stoma, with the result that the jejunal wall is tightly stretched across the artificial orifice, interfering with the entry of alkali. The mere fact of the sudden distension of the jejunum with ejected chyme may also be sufficient stimulus to rapid contraction of this organ, with the same effect.

It should not be forgotten that the presence of an appreciable barium residue at six hours does not necessarily indicate obstruction of the stoma. Sometimes a wrongly placed stoma may have the same effect as shown by Spriggs.[13] Though this might occur in an inactive stomach with a heavy barium meal, it is not incompatible with a comparatively rapid disappearance of a gruel test-meal. It is important to note that the patient of Case 4, though probably an example of this, reports himself as completely cured.

TABLE I.

GROUP A: PATIENTS IN WHOM POST-OPERATIVE ACIDITY HAS FALLEN TO ZERO OR NEARLY ZERO.

Case Number.	Lesion.	Operation.		Result of Radiographic Examination.		Free Acid.		Effect of Operation on Emptying Time.	Duodenum.	After Results.	Remarks.
		Date.	Nature.	Before Operation.	After Operation.	Before Operation.	After Operation.				
12	Duodenal ulcer	7/10/22	Gastro-enterostomy	Orthotonic		70	15	Slow, unaltered	Exclusion	Great improvement, flatulence	Cicatrical stenosis
13	Pyloric ulcer	15/ 9/22	Pólya's	Orthotonic		50	12	Accelerated	Exclusion	Complete cure; can eat anything	Cicatrization evident
14	Duodenal ulcer	10/ 6/22	Gastro-enterostomy			62	0	Accelerated	No exclusion	Much better; weight increased	
15	Duodenal ulcer	10/ 9/22	Gastro-enterostomy	Orthotonic		60	0	Increased	No exclusion	Not known	
16[1]	Gastric ulcer	1916	? Gastro-enterostomy			?	0	Rapid	?	Well for seven years; pain after food recently, relieved by alkali	
17	—	20/ 9/22	Gastro-enterostomy	Atonic ptosed, normal peristalsis, half residue at six hours		21	0	Accelerated	No exclusion	Cured; care required, weight increased	No gastric lesion, pancreas enlarged; pancreatic reactions obtained
18[1]	—	1903	Roux *en-Y*	Orthotonic, vigorous peristalsis, one-sixth residue at six hours, spastic pylorus	Stoma functioning, one-third residue at six hours	?	0	Rapid	?	Pain after meals, flatulence and loss of weight	No gastric lesion; *tabes dorsalis*; gastro-enterostomy twenty years ago; later Y-anastomosis made
19[1]	Duodenal ulcer	27/ 4/22	Gastro-enterostomy			?	8	Rapid	No exclusion	Complete cure	Operation for pyloric obstruction
20[1]	? Pyloric ulcer	10/ 3/22	Gastro-enterostomy		Orthotonic, slight six hour residue, stoma functionating, no pyloric emptying seen	?	0	Normal or longer	Exclusion	No improvement; attacks of great pain and vomiting	
21	*Carcinoma pylori*	20/10/22	Pólya's	Hypotonic		21	0	Accelerated	Exclusion	Complete cure; weight gained	Resection of small carcinoma and Pólya's operation
22[1]	Duodenal ulcer	9/ 2/23	Gastro-enterostomy		Orthotonic, high stoma with small emptying mainly by pylorus	?	9	Rapid	No exclusion	Great improvement	
23[1]	Gastric ulcer	1/ 3/23	Gastro-enterostomy	Ortho-hypotonic, trace of stomach residue at six and twenty-four hours		?	0	Rapid	No exclusion	Complete cure	
24[1]	Gastric ulcer	13/ 2/23	Gastro-enterostomy	Quarter residue at twenty-four hours, organic hour-glass contraction of *pars pylorica* with stasis in lower pouch		?	0	Rapid	No exclusion	Complete cure	Old gastric ulcer with extensive post-operative contraction from operation in 1920
25[1]	Gastric ulcer	7/ 8/22	Gastro-enterostomy and resection of ulcer	Hypotonic, ptosis, six hours' residue		?	0	Rapid	No exclusion	Complete cure	
26[1]	Gastric ulcer perforated	15/ 1/23	Gastro-enterostomy		Orthotonic, stoma functionating, no emptying by pylorus	?	0	Rapid	Exclusion	Complete cure	
27	Gastric ulcer	5/11/23	Gastro-enterostomy	Orthotonic, one-fifth residue		34	0	Accelerated	No exclusion	Not known	

TABLE II.

GROUP B.: PATIENTS IN WHOM OPERATION HAS PRODUCED LITTLE OR NO EFFECT ON THE ACIDITY CHART.

	perforated		enterostomy		no emptying by pylorus						
27	Gastric ulcer	5/11/23	Gastro-enterostomy	Orthotonic, one-fifth residue		34	0	Accelerated	No exclusion	Not known	

TABLE II.

GROUP B.: PATIENTS IN WHOM OPERATION HAS PRODUCED LITTLE OR NO EFFECT ON THE ACIDITY CHART.

Case Number.	Lesion.	Operation.		Result of Radiographic Examination.		Free Acid.		Effect of Operation on Emptying Time.	Duodenum.	After Results.	Remarks.
		Date.	Nature.	Before Operation.	After Operation.	Before Operation.	After Operation.				
1	Duodenal ulcer	10/ 7/22	Pólya's	Good tone		36	75	Increased	Exclusion	Not known	
2	Duodenal ulcer	3/10/22	Gastro-enterostomy	Orthotonic, one-sixth residue at six hours		68	70	Accelerated	No exclusion	Not known	
3	Gastric ulcer	9/10/22	Gastro-enterostomy	Orthotonic, one-third residue	Ortho-hypotonic, stoma functioning, trace residue	45	38	Same	No exclusion	Complete cure	Densely scarred stomach from old partial gastrectomy eighteen months before operation attached to transverse mesocolon
4[1]	Duodenal ulcer	22/ 9/22	Gastro-enterostomy	Hypotonic, one-quarter residue at six hours	Orthotonic, one-sixth residue, no stoma seen, emptying per pylorus	?	43	Rapid	Exclusion	Complete cure, gained nineteen kilcgrams	
5[1]	Pyloric ulcer	23/ 6/20	Pyloroplasty		Orthotonic empty in six hours	?	55	Slow		Cured for three years, symptoms returned lately	
6	Duodenal ulcer	26/ 7/23	Gastro-enterostomy	Atonic, three-quarter residue, active peristalsis	Hypotonic, three-quarter residue, some pyloric emptying	25	58	Same	Exclusion	Only slight improvement, much flatulence and palpitation	Marked pyloric obstruction
7	Duodenal ulcer	20/ 5/22	Gastro-enterostomy	Orthotonic	Ortho-hypotonic and stoma functioning	26	55	Increased	No exclusion	Greatly improved, well on alkalis	
8[1]	Appendicitis	4/ 5/23	Gastro-enterostomy and appendicectomy	Hypotonic, normal evacuation	Hypotonic, spasm of pylorus, one-half residue, no stoma, emptying by pylorus	?	43	Rapid	No exclusion	Unrelieved one month after operation	9/6/23 high acid; 23/7/23 very low acid for one hour then sudden rise to 76
9	Subacute pancreatitis	25/11/22	Gastro-enterostomy	Hypotonic, ptosis and peristalsis not seen, residue one-half at six hours	Hypotonic, residue one-sixth patent pylorus, later stoma was seen functioning, empty in six hours	65	47	Accelerated	No exclusion	Perfectly well till 15/8/23, not so well since	Normal stomach and duodenum greatly thickened head of pancreas with fat necrosis, pancreatic tests yield reactions
10[1]	Duodenal ulcer	11/10/23	Gastro-enterostomy		Orthotonic, stoma functioning, also fair emptying by pylorus	?	32	Slow	No exclusion	Very recent case, well at present	
11[1]	Gastric ulcer	12/11/23	Gastro-enterostomy		Stoma and pylorus both functioning well	?	43	Rapid	No exclusion	Very recent case, but well at present	Perforated gastric ulcer in pyloric region

[1] Only the post-operative acidity chart was obtained.

One further point is important to mention. In the same paper Spriggs has pointed out that in X-ray examinations at one time all food will pass by the stoma and at another time all by the pylorus. Non-function of the stoma cannot, therefore, always be diagnosed by one examination.

From the data presented it would appear that the causes of high acidity after operation in Group (*b*) are as follows:

Cases 1, 2, 4, 5 and 7.—Defective jejunal forces combined with good gastric tone.

Case 3.—Extensive gastric fibrosis and possibly defective jejunal forces.

Case 6.—Pylorus and stoma both nearly closed.

Case 8.—Intermittent spasm of the stoma.

Case 9.—Defective alkali secretion.

Cases 10 and 11.—Operation too recent to give definite result, but well at present. These patients are not included in the general summary of results referred to later under "Conclusions."

Cases 3, 4, 5, 7 and 9.—Completely cured or greatly improved.

Cases 6 and 8.—Failure of treatment.

Cases 1 and 2.—No report of present condition.

Summarizing these results we might say that in the main high gastric acidity after posterior gastro-enterostomy is due to: (i.) Defective jejunal reflux forces, either intrinsic or due to an increase in the forces to be overcome, such as hyper-irritability of the duodenum and upper part of the jejunum, sometimes combined with defective gastric relaxation, tonic or organic. This group may be free of symptoms. (ii.) Obstruction at the stoma, with persistence of symptoms.

From this it is evident that high gastric acidity after gastro-enterostomy is not of itself necessarily of evil import. As the after-results show, some of these patients are quite well and happy. When, however, symptoms accompany the high acidity, obstruction of the stoma and pylorus is suggested and demands investigation. Wyllys Andrews[14] says: "Most of the difficulties after gastro-enterostomy are due to hyperacidity," but it is evident that hyperacidity does not necessarily mean difficulties. There is, however, some danger in these instances of symptomless hyperacidity after operation in the formation of jejunal ulcer. Jejunal ulcer may then be either the cause or the result of high acidity. I need not go into the evidence connecting this condition with high acidity, but that it is one of great importance demanding the investigation of the acidity in all patients after operation is evident if we are to accept the statement of Davis[15] that the condition occurs in 8% of patients subjected to gastro-enterostomy. And he regards this figure as conservative!

There remain only a few more points which have been noted.

The Effect of Exclusion of the Duodenum on Post-Operative Acidity.

In 1921 Sherren[16] stated that exclusion of the duodenum at operation for gastro-enterostomy always led to high post-operative acidity and a higher incidence of jejunal ulcer than where the duodenum was not excluded. He quotes Haberer, who had fourteen jejunal ulcers in seventy-one patients with pyloric exclusion, but only three in two hundred and sixty-five patients without exclusion. Both Guy[6] and Hunter[9] disagree with this as a result of many examinations of acid value before and after operation. They state that exclusion makes no difference to post-operative results. In my own series exclusion of the duodenum has been produced in certain patients either by operation or by cicatricial stenosis. In Group (*b*) there are nine patients after simple gastro-enterostomy with duodenal exclusion in two, and one after Pólya's operation, making three patients with exclusion out of a total of ten in this group. In Group (*a*) are thirteen patients after simple gastro-enterostomy, of whom nine have a patent pylorus and duodenum, three have partial or complete occlusion, and one in whom this point could not be ascertained. In addition there are two patients after Pólya's operation and one after Roux *en-Y* anastomosis operations, making therefore five out of fifteen patients with duodenal exclusion. It is clear then from all this evidence that exclusion of the duodenum has no effect on post-operative acidity.

Emptying Time.

Most observers are agreed that gastro-enterostomy as a rule leads to much more rapid emptying of the stomach, the average time being from one and a quarter to one and a half hours, irrespective of the position of the ulcer, while after partial gastrectomy and Pólya's operation the time is even less, averaging about three-quarters of an hour. Hunter[9] found accelerated emptying in 70% of his patients. My own series shows the same in 68% with the same average time of 1.4 hours in both Groups (*a*) and (*b*), while of three patients submitted to Pólya's operation, in one the time was increased and in two it was reduced to an average of about three-quarters of an hour. The actual diminution of emptying time in Group (*b*) has not been so great, since this group contains a larger proportion of patients with duodenal ulcers in whom the stomachs often empty more quickly before operation than normally. These figures dispose of obstruction at the stoma as a general cause of high acidity in Group (*b*).

Bastedo[17] holds that emptying time is not accelerated in non-obstructive cases, but with him I cannot agree. Guy[6] states that when pain and vomiting had recurred after operation, the stomach took two or more hours to empty. Only one of my patients from whom recent reports have been received, presents these symptoms and his emptying time was about two hours, but of those seven patients whose stomachs take two or more hours to empty three did not reply to my inquiries, three were improved (one only slightly) and one reported absolute cure.

I should add here that I have judged the emptying time by the disappearance of residue from samples withdrawn, whereas other writers judge by the disappearance of the starch reaction. They are not always the same.

Further, as pointed out before, a barium residue at six hours does not necessarily mean that the gruel of a test-meal will be retained for an abnormally long time. Sometimes the reverse occurs.

The Appearance of Bile in the Stomach After Operation.

As is to be expected, bile appears much more frequently and strongly after operation than before. In my series bile was present before operation in the fasting contents in 17% and in the meal samples in 46% of patients, but appeared in 96% after operation [90% of Group (*a*) and 100% of Group (*b*)]. Excluding those who had undergone Pólya's operations, bile was present in 100% of patients after gastro-enterostomy. Guy's[6] findings were similar. Hunter[9] found bile in 90% of fasting contents after operation, but my own series, curiously enough, gives only 54%.

Conclusions.

1. A certain proportion of all patients after gastro-enterostomy show a high acidity, the curve of which is very similar in value and form to that found before operation.

2. In the absence of stenosis at the stoma, this appears to be due to defective reflux forces in the jejunum, perhaps combined with a high gastric and duodenal tone or organic changes preventing gastric relaxation. This group includes the great majority of patients with duodenal ulcer who generally have a good gastric tone. High gastric acidity after operation, therefore, does not necessarily indicate evil, except in so far as it may predispose to jejunal ulceration.

3. Neutralization of gastric contents may also be prevented by some obstruction at the stoma. High acidity with symptoms after operation, therefore, suggests a faulty stoma.

4. Gastro-enterostomy appears to be a device whereby the faulty reflux forces of the jejunum are augmented by the driving forces of the stomach, by making the latter open into the intestine below the ampulla of Vater. By this means chyme projected into the intestine is partly driven into the duodenum, driving pancreatic alkali before it into the upper part of the duodenum, with neutralization of acid there and consequent removal of the cause of pyloric spasm and pain.

5. When the stomach is of low emptying power (atonic, hypotonic or poor peristalsis or obstruction at the stoma) this augmenting power may fail with persistence of symptoms.

6. Of those patients presenting definite gastric or duodenal ulceration, and from whom late reports have been received, operation gave a complete cure in 63%, improvement in 31% and failure in 6% of cases.

7. The main importance of these observations is the light they throw on the working of the gastro-intestinal tract in duodenal ulcer and sthenic dyspepsia, thus giving us a basis for a rational scheme of treatment.

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Reports of Cases.

MECKEL'S DIVERTICULUM WITH AN UMBILICAL POLYP.

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Introductory.

When the umbilical cord is formed in the embryo, the remains of the yolk sac are pushed into the neighbourhood of the placenta where traces of it are frequently to be found at birth.

The vitello-intestinal duct which unites the cavities of the yolk sac and the primitive intestine, becomes a long narrow tube. It is usually occluded at the end of the

sixth week. Its mesodermal portion takes part in the formation of the Wharton's jelly of the cord and its endodermal lining disappears, though Bland Sutton states that traces may be fund at the umbilical end of the cord as late as the fifth month. Umbilical polyps covered with mucous membrane are due to the persistence of this portion of the endoderm of the vitello-intestinal duct.

The intra-abdominal portion usually disappears, but may remain as Meckel's diverticulum. Sometimes it is short and wide, but usually it is long and narrow like an appendix, but without a mesentery. Keith states that on rare occasions a mesentery or the remnants of one crossing the surface of the bowel may be present. It is lined by intestinal mucous membrane, but sometimes the tip contains gastric mucous membrane or an accessory pancreas. It is sometimes the seat of malignant neoplasms and not infrequently the cause in one way or another of intestinal obstruction. It may be attached to the deep surface of the umbilicus with or without an umbilical polyp and may open externally forming a fæcal fistula. At times it becomes inflamed and gives rise to symptoms like those of acute appendicitis. In some cases it causes an umbilical cyst. The condition of septate ileum or congenital absence of a short segment of ileum is said by Bland Sutton to occur at the attachment of the vitello-intestinal duct. Meckel's diverticulum is found in umbilical, inguinal or femoral herniæ as was first described by Littré whose name is given to this type of hernia.

Clinical History.

C.G., a male, *ætatis* two months, a patient of Dr. Shortland, had an umbilical polyp present since the cord separated. The mother had no other living children. The first child was born prematurely at seven and a half months and lived twelve hours. The second child, apparently normal, died at eleven months of gastro-enteritis. C.G., the third child, showed some abnormalities at birth. Labour was precipitate. The *liquor amnii* was brown and turbid, the membranes were thick and gelatinous, the cord was 1.6 centimetres in diameter and extremely hard so that a moderately tight ligature cut through and three loosely applied ligatures were necessary. No typical Wharton's jelly was seen.

On examination the infant was found to be normal except for an umbilical polyp about the size of a terminal phalanx of a finger projecting from the umbilical depression and attached to the bottom by a narrow stalk. It felt firm and was dull red and covered with a purulent exudate.

At operation the umbilical area and the polyp were dissected free and a Meckel's diverticulum found adherent to the deep surface. What seemed to be a single vessel ran from the mesenteric vessels over the surface of the ileum and along the diverticulum to the deep surface of the umbilicus. It was loosely attached to the diverticulum and seemed to give no branches to it. The diverticulum was ligated close to the intestine and removed with the polyp; the base was not invaginated. Cullen (*Surgery, Gynecology and Obstetrics*, September, 1922) described an almost similar case in which the stump of the diverticulum was buried by a purse-string stitch, but became so swollen that it almost occluded the lumen of the bowel and caused obstruction and a fatal termination.

After operation the child (C.G.) slept well, but died rather suddenly about twenty-four hours later without symptoms indicating any cause.

Autopsy Findings.

Autopsy revealed the presence of three congenital bands. One of these was the means of binding the ileum tightly down to the right iliac fossa about twenty-five or thirty centimetres from the ileo-caecal junction. From there the ileum made a loop towards the liver and down to the ileo-caecal valve, both limbs being bound to the ascending colon by two bands close together. The diverticulum had been attached 7.5 centimetres distal to the first band. A volvulus had occurred involving the ileum distal to the first band, the caecum, the ascending and half the transverse colon producing obstruction.

It seemed as though the attached diverticulum had neutralized the effect of the bands and its removal allowed volvulus to occur. There were also several small accessory spleens, but no other congenital abnormalities.

Comment.

The vessel running across the ileum and along the diverticulum is noteworthy. It is the omphalo-mesenteric artery which during early embryonic life supplies the primitive intestine and the yolk sac. At one stage of development it is the largest of the ventral branches of the aorta and surrounds the intestine at the junction with the vitello-intestinal duct with an arterial ring. One of the limbs of the ring—usually the left—atrophies, so that when the artery is present with a diverticulum, it more commonly crosses the right or upper surface of the ileum. The persistent part of the artery becomes the superior mesenteric.

Remnants of the vitelline veins are only exceptionally seen in this situation. The distal ends atrophy early and the central ends take part in the establishment of the portal circulation. The superior mesenteric vein is a secondary channel which has arisen to drain the gut wall alone and has no connexion with the yolk sac.

Pathological Report.

Professor Welsh examined sections through the polyp and reported: "The diverticulum ends blindly at the level of the skin and is lined by normal mucous membrane. The tumour consists of granulation tissue with many giant cells absorbing foreign bodies. The section is identical with one at the University of giant cells absorbing a gauze pad left in the peritoneal cavity. Foreign bodies are present like strands of linen or cotton."

Probably the polyp is a large mass of granulation tissue thrown out to absorb portion of the most proximal of the three ligatures placed on the umbilical cord.

Reviews.

THE CEREBRO-SPINAL FLUID.

IN the second edition of Abraham Levinson's "Cerebro-spinal Fluid in Health and in Disease," the author has included new data with a view to incorporating the results of most recent research and has succeeded in presenting a useful and practical book dealing with this important subject.[1] The hope expressed in the preface to the first edition of "saving the busy practitioner the irksome task of consulting countless sources for information on cerebro-spinal fluid" has certainly been fulfilled though much of the material presented will prove more useful to the laboratory worker than to the general practitioner. The contents are divided into ten chapters with a bibliography at the end of each. The first chapter is historical and surveys the work carried out on cerebro-spinal fluid from the time of Hippocrates to the present day. In the second the author deals with anatomy and physiology. In the chapter on the collection of cerebro-spinal fluid detailed instructions for the carrying out of lumbar puncture, ventricular puncture and puncture of the cistern are given with indications for and contra-indications to these operations. The chapter on the methods of examination of cerebro-spinal fluid for diagnostic purposes is both simple and practical. The details of the technique for the various tests are clearly presented, difficulties likely to be encountered are noted and methods of avoiding them suggested. In a chapter so well arranged and presented the section devoted to the performance of the Wassermann reaction is the only disappointing feature. Too little detail has been given for the satisfactory carrying out of this important and difficult test, too much for the presentation of those factors in the Wassermann reaction which have a special bearing on its application to cerebro-spinal fluid. The final chapters deal with the fluid in various diseases and with intra-spinal treatment. The book can be strongly recommended to the practitioner and the laboratory worker, as in the production of a concise and practical work on cerebro-spinal fluid a long-felt want has been supplied.

[1] "Cerebro-spinal Fluid in Health and in Disease," by Abraham Levinson, B.S., M.D., with a Foreword by Ludvig Hektoen, M.D.; Second Edition, Thoroughly Revised; 1923. St. Louis, U.S.A.: C. V. Mosby Company; Demy 8vo., pp. 267, with 69 illustrations. Price: $5.00.

The Medical Journal of Australia

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The Coordination of the Health Services.

Among the many lessons of the war none was of more far-reaching importance than that of the co-ordinated action of the several branches of the medical services in the prevention of physical unfitness. The experience born of necessity and nurtured by the single idea of keeping the front line full has demonstrated that it is possible to organize a campaign of prophylaxis on an immense scale and to fit into this scheme individuals and unfits widely separated and scarcely aware of each other's existence. The reduction almost to vanishing point of enteric fever is often quoted as the supreme achievement of the several army medical corps. It certainly was a masterly work of great importance, but it represents but one link in that immensely powerful chain of prevention which was forged in the early years of the war. The real achievement lay in the fact that the armies of the world were able to apply the knowledge previously gained by patient research together with fresh information gathered during the course of the struggle with such uniform success.

In civilian life we are only just beginning to realize that a similar coordinated effort to raise the general standard of fitness of the community and to reduce the incidence and mortality of many diseases is within the bounds of possibility. Hitherto we have been content to watch the waxing and waning of epidemics, to tinker with drainage and water supply systems and to argue about the milk supply. The care of the health of the people is placed in the hands of many independent authorities and each has been expected to control disease and enhance well-being at the lowest possible cost. The public has not been in earnest in its determination to apply modern prophylactic measures on a large scale and in a scientific spirit. Enteric fever, tuberculosis, diphtheria, syphilis and all the other common infective diseases have been tolerated with a complacency worthy of the fatalistic Turk. It is true that sanitation, the elimination of slavery from labour and the provision of facilities for health-giving out-door recreation among many other factors have led to a sensible reduction in the incidence and mortality of some of these diseases. Almost everyone of the speakers at the final discussion at the Congress on tuberculosis referred to the reduction in the incidence of tuberculosis as hopelessly inadequate. Congress resolved to seek fresh facts. The nations during the war set to work armed with the available knowledge and achieved an extraordinary degree of success. What is needed to hasten the practical development of communal prophylaxis? Why is so little progress registered year by year? Is it not because of the lack of coordination and determination? In the war the order, universally issued and as universally respected, was to maintain man power at all costs. Man power means healthy bodies and healthy minds. At present we have one authority held to be responsible in each State for the establishment and maintenance of hygienic conditions, another authority charged with the care of the health of school children, another authority entrusted with the task of looking after the insane. There is no authority whose duty it is to organize a campaign for the maintenance of the health of every individual from the time of conception to the time of advanced age. It is nobody's business to attack every possible source of disease and defect before the innumerable noxes wreak their vengeance on humanity. What is wanted is a general recognition of the possibility of tracing disease to its ultimate cause. If every man and every woman in the community recognized this, there would be a demand for a general warfare on disease, cost what it might. In the next place it is essential that the public health services, the school medical services, the lunacy services, the infant welfare organizations, the maternity institutions and the thousand and one other activities that have or should have the prime motive of prevention, should be coordinated under one authority. The order of the day should be simultaneous attack all along the line. Independent and isolated action should be replaced by concerted advance. The idea governing these offensives should be expansive and

ambitious. There should be no hesitation, no half measures, no compromise. The action should be to trace existing diseases to their sources and to stifle them when the sources are discovered. With co-ordination of the medical services there would have to be extension in two directions. The number of trained experts working in the services would have to be greatly augmented. Simultaneously the aid of the whole of the practising profession would have to be enlisted, just as it was in the war. Not one disease should be attacked at a time, but all, so that the civil army of the medical profession might maintain man power. But the first step in real prophylaxis must be the weaving together of all the independent services and authorities, so that every onslaught may be effective.

Current Comment.

THE GERMICIDAL ACTION OF THE GASTRIC JUICE.

IT is sometimes forgotten that the specific function of the stomach is two-fold, digestive and antiseptic. The digestive function is concerned with the chemical action of the gastric secretion on the ingested food either in rendering it fit for absorption or in preparing it for the action of the secretions of the duodenum and small bowel. Its antiseptic function consists in the partial destruction of organisms which have entered the stomach from the mouth or naso-pharynx or have been introduced as part of the food itself. The obvious nature of the former function is often allowed to overshadow the latter. Bunge held that the antiseptic action of the gastric juice was as important as its digestive function and that it was due to the presence of hydrochloric acid. Among those who have in latter years discussed the subject, is Hurst. He drew attention to the association of *achylia gastrica* with appendicitis and cholecystitis. He referred to the findings of Bonar that 33% of sixty-five patients operated on for appendicitis had little or no free hydrochloric acid in their stomachs throughout the course of a fractional test meal. Bonar found that hyperchlorhydria was present in 55% of his patients. Hurst expressed the opinion that the hyperchlorhydria in these individuals was the result of the appendicitis and added that he could not see how the achlorhydria could also be secondary. He thought it more probable that primary *achylia gastrica* predisposed to chronic appendicitis by helping to maintain or to aggravate an hæmatogenous infection "by permitting the access into the lumen of the appendix of excess of oral streptococci which in a normal individual would have been destroyed to a large extent by the gastric juice." He expressed very much the same view in regard to cholecystitis.

Dr. F. A. Knott has recently discussed this question.[1] He states that the existence of the antiseptic defence of the gastric juice seems to be established clinically, but that it is still undecided as to its full mechanism and as to which strains can most easily pass the gastric secretion. Dr. Knott draws attention to the fact that the conditions of experimentation are not exactly similar to those occurring in the stomach. The period of time to which he exposed the bacteria in his experiments, was twenty minutes. In normal digestion some of the stomach contents leave the stomach before this time, but this is not so with the greater part of the contents which are in contact with the gastric juice for a longer period. At the same time in experiments of this nature an end point is reached when every organism has been killed. In the stomach shorter exposures will kill considerable numbers of the organisms and the vitality of many others will be seriously impaired. In testing the germicidal power of combined hydrochloric acid dilute acid was added slowly to milk and to nutrient broth previously inoculated with *Bacillus typhosus* until protein combination was complete; as more acid was added this was recognized by means of Gunzberg's reagent. Samples taken at intervals were incubated for twenty minutes. No killing of bacteria could be detected until free acid was definitely present in the original mixture. Dr. Knott concludes that the acid in protein combination has no germicidal power. From the fact that a 25% solution of sodium chloride did not kill a twelve hour broth culture of *Bacillus coli communis* in twenty minutes, he concludes that the very smaller amounts of inorganically combined hydrochloric acid found in the gastric juice cannot have any appreciable lethal power towards bacteria. In order to test the effect of free hydrochloric acid a series of tests was made with dilute acid in normal saline solution. It was found that spore-bearing organisms easily survived an 0.3% solution of acid or even a stronger solution. Resistant strains of *Bacillus coli* were killed by about 0.3% to 0.4% of acid. Bacilli of the typhoid-dysentery groups and the staphylococci were killed by about 0.15% solution. Streptococci, diphtheria bacilli and other delicately growing organisms were killed by 0.02% to 0.03% solutions. The presence of a parallel action in gastric juice was then confirmed by replacing the saline solution by a definitely acid filtrate from a test meal. The filtrate was required to be sterile and to have no peptic activity. The free hydrochloric acid was neutralized by a decinormal solution of caustic soda and fresh acid was added to bring the free acid strengths to the same levels as before. The results which it is unnecessary to quote in detail, were very similar to those previously obtained. Dr. Knott states that the free hydrochloric acid in the gastric juice must have a definitely selective as well as a germicidal activity. As a result of another experiment he concludes that

[1] *Guy's Hospital Reports*, October, 1923.

the peptic activity of the gastric juice has of itself over short periods no germicidal powers comparable with those of free acid.

Dr. Knott concludes that as far as the vigorous killing of bacteria is concerned, the only thing to be considered in the gastric contents is the percentage of hydrochloric acid. It is evident that spore-bearing bacteria and the more resistant coliform bacilli can readily survive passage through the average gastric juice. The more delicate organisms of the typhoid-dysentery groups and the staphylococci will survive only if the free hydrochloric acid tends to be low or the emptying of the stomach tends to be rapid. The streptococci, diphtheria bacilli, micrococci of the catarrhalis group and other readily killed organisms will leave the stomach in the living state only if the quantity of free hydrochloric acid is extremely low or when a condition approaching achlorhydria exists. Dr. Knott claims that his results appear to be in accord with the opinions of other observers that clinical conditions arising from intestinal infection with pathogenic organisms and absorption of their toxins may arise from deficiency of free hydrochloric acid in the gastric secretion. This probably is so; but it must be remembered that although opinions very often form a good working hypothesis, they must be proven before they can be accepted as facts. One practical point as an outcome of Dr. Knott's work is his suggestion that if hydrochloric acid be given by month to remedy a deficiency in the gastric juice, it should be given alone and not be mixed with proteins such as those of milk.

HOSPITAL STANDARDIZATION.

The work undertaken by the American College of Surgeons in regard to reviewing, organizing and standardizing the work in hospitals has been referred to on more than one occasion in these columns. It was in 1915 that a start was made and since that time progress has been steady and continuous. The history of the movement and the standards laid down together with the annual results were discussed in detail in our issue of May 12, 1923. The aim of the whole scheme may be defined in a few words as the establishment and maintenance in hospitals of an organized personnel working as a team in the interest of the patient. The demand is made that the staff shall be provided with the necessary facilities for scientific diagnosis and treatment and that these facilities shall be used to the best possible advantage. No interference with the domestic arrangements of an institution or with the initiative of those controlling it is suggested. A standard of minimum requirement is set up and is regarded as fundamental. Approval is not given to an institution failing to reach the standard. The requirements are summarized under three headings: the organization of the medical staff, the keeping of accurate case records and provision of adequate diagnostic and therapeutic facilities.

The American College of Surgeons has recently issued its hospital standardization report for 1923; there is a further increase in the number of hospitals which have attained the requisite standard.[1] In 1922, of 812 hospitals of 100 beds and over surveyed 677 were approved as having attained the standard. In 1923, of 870 hospitals 749 were approved. In 1922, 812 hospitals of between 50 and 100 beds were surveyed and 335 attained the standard; in 1923, of 916 surveyed the number was 427. Adding the two totals for 1923 together it will be seen that 1,786 hospitals were surveyed in Canada and the United States. Of these 1,176 or 66.9% were approved. It is claimed that the increase in numbers shows conclusively that both the medical profession and the public are profoundly interested in the programme of hospital betterment. Four results are claimed for standardization. In the first place it is claimed that the patient's stay in hospital has been shortened, secondly incompetent or unnecessary surgery has been largely prevented, the number of complications and infections has been lessened and the hospital death rate has been lowered. In regard to the lowering of the death rate some interesting figures are quoted. It is stated that the average hospital death rate had generally been from thirty, forty and in some instances as high as fifty per thousand patients treated. The rate has been declining in certain institutions which have been working strictly along the lines laid down for hospital standardization. In some instances the death rate has fallen to thirty, twenty or even less per thousand patients. It is claimed that these results have been accomplished through the sincere, honest and conscientious application of the principles laid down by the American College of Surgeons. Another statement in the report is worthy of mention. This is that the organization of the medical staff of a hospital acts as a stimulus to better organization throughout the institution. It can easily be understood that the adoption of better organization and a higher standard by the medical staff could not fail to react in any but a beneficial manner on the nursing staff, students and attendants. Example is always better than precept. Consideration is now being given to the standardization of hospitals of less than fifty beds.

Hospital standardization is an ideal to be kept constantly in mind in regard to Australian hospitals. This does not mean that the American system is to be accepted *in toto*. Allowances will necessarily have to be made in order to conform with Australian conditions. The honorary staffs of the larger metropolitan hospitals can initiate the work in a small way by endeavouring to control their results as a team. If the boards of control can be persuaded that beneficial results can be obtained by some form of standardization and the subscribers to hospitals are once made to realize that more satisfactory treatment of patients would result, an Australian scheme of standardization will be initiated. The whole scheme may be idealistic, as opponents have so often stated, but even if that be so, nothing but good can result from striving to attain the ideal.

[1] *Surgery, Gynecology and Obstetrics*, November, 1923.

Abstracts from Current Medical Literature.

THERAPEUTICS.

Digitalis.

G. A. SUTHERLAND (*The Lancet*, December 8, 1923) asserts that neither digitalis nor any other known drug acts directly and beneficially on the heart. The outstanding function of the heart, namely its pump-like action for the maintenance of the circulation, cannot be improved or increased by the direct action of drugs. Yet such is the teaching of those pharmacologists who write text-books for credulous students. The virtue of digitalis lies in its influence on disturbances of the cardiac action, whether in rate or rhythm or both. For example, in auricular fibrillation all the physician can do is to reduce the excessive number of ventricular beats. He can then rely on the muscular tone and the contractile power of the heart to do the rest. The vast majority of affections of the rate and rhythm arise in the supra-ventricular tissues, that is those parts of the heart which lie above the purely contractile tissues of the right and left ventricles. They are the conducting tissues of the heart and include the sino-auricular node, the auricles, the auriculo-ventricular node and the auriculo-ventricular bundle. The action of digitalis is to stimulate the vagus, the great controlling nerve of the heart. The result is a depression or inhibition of the activity of the supra-ventricular tissues, manifested by a slower production of impulses at the sino-auricular node (the "pace-maker") and by a slower rate of progress through the conducting tissues, so that the *a.—v.* interval is lengthened. In auricular flutter and fibrillation the sino-auricular node is not in action, since the heart beat starts elsewhere. In these conditions the vagus nerve acts on some other part of the conducting tissues, especially the auriculo-ventricular node and bundle, diminishing their rate of conduction. Such an action on the vagus by digitalis results in a slowing of the ventricular rate. Hence the aphorism: "No slowing of the ventricular rate, no benefit from digitalis." In the treatment of disease of the pericardium, myocardium and endocardium, as such, digitalis plays no part. No variety of heart disease, *per se*, is an indication for digitalis, but in all forms of heart disease characterized by heart failure and accompanied by a rapid ventricular action digitalis may be useful. The drug is equally serviceable whether the valvular lesions be mitral regurgitation or mitral stenosis or aortic regurgitation or a combination of these, provided that it is indicated for other reasons. A regular cardiac action is no contra-indication to the use of digitalis. Beneficial effects are more strikingly manifested in diseases of rheumatic origin, possibly because the cardiac tissues have been so altered by the rheumatic infection that they are more susceptible to the inhibitory action of the vagus when stimulated by digitalis. Digitalis is of no value in extra-cardiac diseases which react on the heart. Active toxæmia interferes with the action of the drug on the heart, as, for example, alcoholism and Graves's disease. But when the active symptoms have subsided and toxæmia no longer exists, damage to the heart revealed by auricular fibrillation or tachycardia may be vastly benefited by digitalis. Similarly in acute pyrexial conditions, digitalis does not slow the heart. Repeated disappointments have led many to abandon the drug in the treatment of pneumonia. Finally, it should be remembered that children benefit from digitalis as much as do adults, but they require much larger doses in proportion to their size.

Quinidine.

C. C. WOLFERTH (*Annals of Clinical Medicine*, September, 1923) summarizes the present status of quinidine therapy. He states that the drug depresses contractility, conductivity, stimulus production and excitability and prolongs the refractory period. Paresis of vagus action is caused by appropriate dosage. Quinidine is excreted mainly in the urine; excretion begins quickly and is greatest during the first twenty-four hours. The principal effects of the drug last one or two days. The author states that the action in terminating auricular fibrillation and flutter might be by the closure of the gap in the circus movement as described by Lewis. Quindine lengthens the refractory period of heart muscle, therefore the oncoming wave in circus movement travelling into tissue recently stimulated would find it still in a refractory stage. Reports indicate that with very little selection of patients auricular fibrillation can be abolished in nearly 50% of those suffering with that form of irregularity of the heart beat. Recent instances are most favourable for treatment. When conspicuous heart failure is present, quinidine is dangerous. Quinidine sulphate 0.2 gramme is given first and the dose increased as required up to one gramme thrice daily; the effects must be carefully observed all the time. Rest is essential for a time after restoration of normal rhythm. The majority of patients relapse sooner or later. Quinidine is sometimes useful in paroxysmal tachycardia and in extra systoles a more regular action of the heart may occasionally be obtained. The drug appears to be harmful in simple tachycardia and in hyperthyreoidism; the patients usually feel worse as a result of its use.

Bismuth.

B. LUCKE AND J. V. KLANDER (*Journal of Pharmacology and Experimental Therapeutics*, June, 1923) give the results of their investigation into the histological changes produced by injections of bismuth compounds into rabbits. The following preparations were injected intravenously or intramuscularly: sodium and potassium tartro-bismuthate (in aqueous solution and oily suspension), potassium tartro-bismuthate (in aqueous solution) and bismuth trioxide (in oily suspension). All the animals died within sixteen days. Autopsies were made soon after death and sections of brain, heart, lung, liver, kidneys, adrenals and spleen were examined. The main lesions occurred in liver and kidneys. The liver manifested cloudy swelling, some nuclear degeneration, fat droplets and occasional small foci of necrosis flooded with erythrocytes. The kidneys were the site of more definite changes, namely degeneration of the epithelium of the convoluted tubules (cloudy swellings, necrosis and calcification) and the presence of agglutinated and sometimes hyalinized masses of erythrocytes in the glomerular capillaries. In other organs mild cloudy swelling only was found. The toxic effects of bismuth preparations used in the treatment of syphilis, therefore, appear to be most noticeable in the kidneys and liver.

Digitalis.

W. D. REID (*The Journal of the American Medical Association*, August 11, 1923) discusses some toxic effects of digitalis. He studied a number of patients to whom digitalis was administered. Thirteen of these patients suffered with symptoms due to the toxic effects of digitalis; coupled rhythm, partial heart block, the onset of auricular fibrillation and paroxysmal tachycardia originating in the ventricle were the principal cardiac disturbances noted. It was found that in the thirteen instances mentioned the dosage of digitalis was usually far in excess of that needed to produce the maximum effect according to the Eggleston method of administration. According to this method one cubic centimetre of the tincture or 0.1 gramme of the leaf for each 4.5 kilograms body weight of the patient is sufficient. The author suggests that if signs or symptoms appear that may be due to toxic effects of the drug and if calculation shows that the amount already given is near or above the Eggleston dosage, additional digitalis should be administered cautiously, if at all. In order to avoid error, the tincture of digitalis used by the author was carefully tested for potency and was also used on nine patients suffering from heart failure in whom no toxic effect was observed, but in whom the therapeutic effect was satisfactory. In estimating dosage the author calculated that a patient disposes of about 1.32 cubic centimetres of the tincture of digitalis *per diem* and he therefore subtracted 1.32 cubic centimetres for each day on which the drug was given, in estimating the total dosage. For instance, a woman weighing sixty kilograms (one hundred and thirty-two pounds) was given four cubic centi-

metres of tincture of digitalis a day for nine days. The total amount given was thirty-six cubic centimetres. After subtracting twelve cubic centimetres for elimination, twenty-four cubic centimetres remain in the patient at the end of nine days. By this date she had developed coupled rhythm. This amount is equal to 181% of 13.2 cubic centimetres which would be the Eggleston dosage for a patient weighing sixty kilograms.

UROLOGY.

Metastatic Gonorrhœa.

J. E. McDOWELL (*The Urologic and Cutaneous Review*, June, 1923) considers that the treatment of metastatic gonorrhœal conditions with vaccines and sera has not stood the test of time. More useful results come from the intelligent use of colloids, metal and non-metal. The method is still largely experimental, however, and too much should not be expected. The question of dosage, for instance, is hardly settled. The writer has greatly increased the doses and decreased the intervals which were described originally by McDonagh. Results have proved the wisdom of this procedure. The most rapid effects seen are in the acute varieties which appear to be going on to suppuration. Often after the second injection of a metal such as manganese or "Trimine" there is a decided improvement. The more chronic varieties need several injections of the non-metal "Intramine" before administration of "Trimine." The efficiency of "Intramine" is greatly increased by the preliminary intravenous injection of colloidal iodine in twenty cubic centimetre doses every other day for a fortnight. In chronic conditions "Intramine" is injected in three cubic centimetre doses for three to five injections at intervals of three to six days. The intervals are regulated by the reactions and the condition of the patient. The treatment is terminated with several injections of "Trimine" in one to two cubic centimetre doses. Even if only slight results are obtained, a similar course should be repeated after a month's rest.

Unilateral Fused Kidney.

W. D. BIEBERBACH (*The Urologic and Cutaneous Review*, January, 1924) in reporting an instance of unilateral fused kidney, states that it is one of the most unusual forms of congenital renal anomaly. Prior to the introduction of the cystoscope these kidney defects were recognized only at the time of operation or autopsy. About seventy instances of unilateral fused kidney have been reported. It was found to occur once in every eight thousand patients. In contrast with this the author states that horseshoe kidney occurs in about one among every thousand patients. Unilateral fused kidney is seen twice as often in the male and is found more frequently on the left side. The majority of fused kidneys are more liable to disease on account of their position and they very often are affected by hæmaturia or pyuria. The patient reported by the author was a man twenty-seven years of age. The kidney of this patient was apparently not the seat of pathological change. The symptomatology is very often vague, especially if the kidney is free from disease. The author's patient did not complain or notice any symptom due to the defect until he was twenty-seven years of age. Up to this time he had been a hairdresser. When he changed his occupation to that of an automobile mechanic, the continual bending caused trauma of the kidney and led to the complaint of pain in the lower quadrant of the abdomen. As the defect was on the right side a diagnosis of appendicitis was made and appendicectomy was performed. No relief was afforded and the pain returned on the resumption of work. Pyelography was undertaken and revealed that the right ureter turned sharply outwards from the ureteral orifice and entered a long narrow pelvis with its centre at the level of the iliac crest. The left turned sharply inwards, crossed the sacrum and entered a pelvis at the level of the fourth lumbar vertebra. The author states that the condition was probably the result of one renal bud being given off higher up than the other from the Wolffian duct during embryonic life and crossing the middle line, forming a bridge and then joining the lower pole of one kidney with the upper pole of the other. The author claims that it is essential that an early diagnosis should be made. It is important that a patient should be acquainted with the presence of a defect so that his living and occupation may be regulated to protect the kidney.

Perineal and Suprapubic Prostatectomy.

R. V. DAY (*California State Journal of Medicine*, September, 1923) summarizes the present position in regard to perineal and suprapubic methods of prostatectomy and gives the special indications for each. When each operation is performed in the most skilful manner and after the most careful preparation, perineal prostatectomy gives a slightly less mortality. On the other hand, it is quite certain that the following disadvantages of the perineal operation exist and occur even with the best operators: (i.) Incomplete removal; (ii.) some degree of incontinence of urine; (iii.) decrease or loss of the sexual power and (iv.) recto-urethral fistula. Incontinence is more apt to follow one of the newer modifications of Young's method (Hinman, Geraghty) when enucleation *en masse* is practised from the perineum. The risk of operation in about 30% of all candidates for prostatectomy is considerable. In dealing with these persons the mortality rate as a result of the suprapubic operation is at least twice as great as that following the perineal method, so the latter should be chosen and the surgeon should be prepared to experience occasionally a bad functional result. The remaining 70% have enough vitality to withstand the suprapubic operation which is more radical and precise and preserves good sphincter control and sexual power. Should a bad structural result occur after the high operation it can be corrected; if after the low, it is most often irremediable.

Treatment of Bladder Tumours.

H. H. YOUNG AND W. W. SCOTT (*New York Medical Journal and Medical Record*, September 5, 1923) sum up the results obtained in the treatment of all patients suffering from bladder tumours at the Brady Urological Institute. These number in all three hundred and eighty persons and the conditions comprised both innocent and malignant bladder growths. Fulguration through the cystoscope is the most suitable method in dealing with papillomata, but when the tumours are large, radium aids in their more rapid disappearance. The use of radium is further indicated by the fact that all vesical papillomata are potentially malignant. In the treatment of malignant papillomata small repeated applications of radium by means of a special cystoscope held in position by a clamp for an hour every day or two are most valuable. The action of radium should be reinforced by fulguration. When the malignant tumour is extensive or definitely infiltrating it should be attacked by the suprapubic route. Tumours of the vertex and upper anterior, lateral and posterior walls are suitable for resection with a wide surrounding zone of healthy bladder wall. In less favourable situations there seems to be a brighter outlook in the future by the employment of radium implantation associated with deep burning either with the actual cautery or by fulguration. The authors conclude that the gloomy outlook which was held as to the curability of bladder tumours, has passed.

Inflammation of the Epididymis.

A. R. STEVENS (*The Journal of Urology*, July, 1923) puts forward some general considerations in the differentiation of tuberculous from pyogenic (non-gonococcal and non-syphilitic) epididymitis. The presence of double epididymitis slightly favours a diagnosis of tuberculosis. A history of a previous orchidectomy or epididymectomy is almost always an indication of the presence of tuberculosis. A scrotal sinus of over a month's duration is probably tuberculous. Tuberculosis elsewhere in the body is an indication of genital tuberculosis in over 90% of persons so affected. In the diagnosis of conditions of over one month's duration help may be derived from rectal examination. The older the lesion, the more does involvement of the prostate and vesicles point to tuberculosis. Finally, it must be emphasized that simple inflammatory changes in the epididymis may last as long as tuberculosis.

British Medical Association News.

MEDICO-POLITICAL.

A MEETING OF THE FEDERAL COMMITTEE OF THE BRITISH MEDICAL ASSOCIATION IN AUSTRALIA was held at the Medical Society Hall, East Melbourne, on February 26 and 27, 1924, MR. G. A. SYME, CHAIRMAN, in the chair.

Representatives.

The following representatives of the several Branches of the British Medical Association in Australia attended:

New South Wales Branch: DR. R. H. TODD, DR. J. ADAM DICK, C.M.G..
Queensland Branch: DR. W. N. ROBERTSON, C.B.E., DR. J. LOCKHART GIBSON.
South Australian Branch: DR. H. S. NEWLAND, D.S.O., C.B.E., DR. B. SMEATON.
Victorian Branch: MR. G. A. SYME, DR. R. H. FETHERSTON.

Apologies for non-attendance were received from DR. GREGORY SPROTT and DR. D. H. E. LINES, representatives of the Tasmanian Branch.

Representatives of the Branches for 1924.

The return of representatives of the Branches elected as members of the Federal Committee for the year 1924 was received as follows: New South Wales Branch, DR. J. ADAM DICK, C.M.G. and DR. R. H. TODD; Queensland Branch, DR. J. LOCKHART GIBSON and DR. W. N. ROBERTSON, C.B.E.; South Australian Branch, DR. F. S. HONE (resigned), DR. H. S. NEWLAND, D.S.O., C.B.E. and DR. B. SMEATON; Tasmanian Branch, DR. D. H. E. LINES and DR. GREGORY SPROTT; Victorian Branch, DR. R. H. FETHERSTON and MR. G. A. SYME; Western Australian Branch, DR. F. A. HADLEY and DR. W. P. SEED.

Officers.

MR. G. A. SYME was re-elected Chairman of the Federal Committee for the ensuing year and DR. W. N. ROBERTSON, Vice-Chairman. DR. R. H. TODD was elected Honorary Secretary and Honorary Treasurer and DR. W. H. CRAGO was appointed Honorary Auditor.

British Medical Association—New Premises.

After the correspondence had been dealt with, it was resolved that a letter should be sent to the Council congratulating it on the successful acquisition of new premises at Bloomsbury for the headquarters of the British Medical Association.

Australasian Medical Congress (British Medical Association)—First Session.

A number of resolutions passed by sectional meetings at the Session held in Melbourne in November, 1923, which had been forwarded by the Executive Committee, were considered.

The Section of Medicine urged that the Federal Government should do all that it possibly could to reduce the cost of "Insulin." In view, however, of the fact that since the Congress the price had been reduced to three halfpence per unit, no action was taken.

The Section of Preventive Medicine and Tropical Hygiene had five proposals. The first was a plea for the fullest recognition by the Commonwealth government of the principles of preventive medicine and its consideration led to the following resolution being passed on the motion of Dr. H. S. Newland, seconded by Dr. J. Lockhart Gibson, namely:

> That, in view of the supreme importance at this period of active growth of the Commonwealth of insuring that the principles of preventive medicine and public health shall be universally applied on the most modern and scientific lines, the Federal Committee respectfully requests the Commonwealth Government to institute a public inquiry by experts with a view to the necessary Commonwealth and State legislation being passed.

The second which recommended the inauguration by State health departments of annual post-graduate courses of instruction for medical officers of health, was allowed to stand over until the larger question, to be considered later on by the Committee, of the cooperation of the medical profession with the Commonwealth Department of Health, had been dealt with. The third aimed at establishing a more intimate association of the Branches of the Association and the public health administration and resulted in a motion by Dr. B. Smeaton, seconded by Dr. H. S. Newland, being carried, namely:

> That it be a recommendation to each Branch to take steps to have a full-time government medical officer engaged in public health work as a member of the Council.

The fourth met with the approval of the Committee and was put into the form of a resolution moved by Dr. R. H. Fetherston and seconded by Dr. J. Lockhart Gibson and carried, as follows:

> That having regard to the fact that it is established that tuberculosis in cattle is an important source of tubercular infection in children the Federal Committee urges upon the State Governments the need of a reasonable scheme of compensation for cattle destroyed because of reaction to the tuberculin test.

In regard to the fourth which requested that the Minister for Home Affairs should arrange for the classification of occupation and the collection of statistical data as to occupational mortality, no action was taken.

The Section of Neurology and Psychiatry reaffirmed a resolution passed by the same Section at the Brisbane Congress of 1920 urging the necessity for teaching bodies to provide instruction in medical psychology to all medical students and the importance of the establishment of psychiatric clinics in all large hospitals. In view, however, of the steps recently taken by the universities to do what was proposed, it was thought that action on the part of the Committee was not called for.

The Section of Radiology and Medical Electricity was responsible for three propositions. The first was a protest against any further increase in customs duty on X-ray apparatus and material and a request for the abolition of duty on Coolidge tubes and duplitized films. The Committee decided to make the necessary representations to the Government. The second was a claim that it should be declared unethical for a medical attendant to refer his patients to a lay radiologist either for X-ray diagnosis or for treatment. The Committee decided to invite the Branches to consider the question with a view to an appropriate rule being made, if thought to be necessary. In regard to the third, under which a committee of radiologists was appointed to consider the formation of a society of radiologists and scientists interested in radiology, to be affiliated with the British Association of Radiology and Physio-therapy, the Federal Committee thought that the objects of the Section would be better served if the members of the Association practising radioology and medical electricity were to form sections for the study of their special subject under the rules of their respective Branches.

The Section of Otology urged that the Australian universities should grant diplomas in oto-rhino-laryngology. The Committee was in accord with the proposal and thought that it should be extended so as to apply also to other special branches of medical knowledge. Following up the suggestion of the Section the Committee resolved, on the motion of Dr. W. N. Robertson, seconded by Dr. J. Lockhart Gibson:

> That the Federal Committee recommends to the governing bodies of the Australian universities that they consider the advisability of instituting post-graduate courses with a view to granting diplomas in various specialties.

The Executive Committee of Congress had forwarded also a number of recommendations made by the Committee of Secretaries of Sections. They were considered *seriatim* and resolutions in respect of them were passed as follows:

(i.) On the motion of Dr. R. H. Todd, seconded by Dr. H. S. Newland:

> That "Suggestions for the Organization of a Session of the Australasian Medical Congress (British Medical Association)" be drafted for submission to the next meeting; and that provision be made therein for a "Committee of the Secretaries of Sections," the local vice-presidents of sections being authorized to act on the Committee.

(ii.) On the motion of Dr. R. H. Fetherston, seconded by Dr. J. Lockhart Gibson:

> That a meeting of the Federal Committee be held the day after the conclusion of each session of Congress to consider resolutions sent on by the various sections.

(iii.) On the motion of Dr. W. N. Robertson, seconded by Dr. H. S. Newland:

> That it be the duty of the Honorary Secretary of the Federal Committee to keep all possibly useful papers and materials from previous congresses to facilitate the organization of future congresses.

Some other resolutions were thought to be impracticable and they were passed over.

It was noted that the final meeting of the General Committee of Congress, required by the Regulations (Regulation 16), had not yet been held. An interim financial statement, however, prepared by the Honorary Treasurer of Congress, was submitted for the information of the Committee.

A letter from the Editor of THE MEDICAL JOURNAL OF AUSTRALIA was read in reference to the publication of the Transactions of Congress. It drew attention to the fact that the papers and discussions in the First Session had aggregated two million words or three times the amount of the matter constituting the Transactions of the 1914 (Auckland) or 1920 (Brisbane) Congresses. It referred also to the strain it had been on the Journal to comply with the wish of the Executive Committee that the publication of the Transactions should follow the Session with as little interval as possible. It submitted recommendations for regulating the number of hours each day a section should sit, and the number and length of the papers to be taken; for limiting the length of speakers' remarks and for speakers to hand in a written summary of their contributions to the discussion; also for the setting up of the papers in type before the opening of Congress. The object of the suggestions was to reduce the bulk of the Transactions and to facilitate their publication without delay and without excessive cost. After careful consideration of the proposals it was left to the Honorary Secretary to embody them, with certain exceptions, in the draft suggestions for the organization of a session to be submitted at the next meeting.

The place and date for the Second Session of Congress were next dealt with. An invitation was received from the New South Wales Branch to arrange for the Second Session to be held in Sydney in 1925 or 1926, as might be determined. It was understood that the Western Australian Branch had contemplated inviting the Committee to arrange for the Second Session to be in Perth, but was prevented from doing so owing to the removal of the temporary University buildings and the consequent absence of accommodation. It was resolved on the motion of Dr. W. N. Robertson, seconded by Dr. H. S. Newland:

> That the Second Session of Congress be held in the year 1926.

On the motion of Dr. R. H. Todd, seconded by Dr. H. S. Newland, it was resolved:

> That the question of determining at what place the Second Session of Congress shall be held stand over until the next meeting of the Committee.

Incorporation of Australian Branches.

Letters were read addressed to the Medical Secretary, Dr. Alfred Cox, and the Solicitor, Mr. W. E. Hempson, of the British Medical Association, forwarding to them in accordance with the directions of the Committee given at its meeting in November last, the Draft Model Memorandum and Articles of Association for use by any Branch in Australia proposing to incorporate. The letter to the Medical Secretary requested that the Draft might, if possible, be submitted to the Council for its approval at its meeting on February 13, 1924. A cablegram, in reply, dated February 15, was read advising that the approval of the Council had been given to the Draft and authorising the Committee to supply the Model to the Branches concerned so that they could proceed forthwith to take steps to incorporate if they wished to do so.

Medical Benefit for Widows of Deceased Soldiers.

At the last meeting of the Committee approval was given to a proposal of the Repatriation Commission to arrange with the friendly society lodges to provide medical benefit for the widows and orphans of soldiers whose death was due to war service and to the widowed mothers of such deceased unmarried soldiers. Correspondence with the Commission and with the Branches was read and it was understood that the arrangements were in progress.

Examination of Recruits for Air Force.

A letter was read from the Secretary of the Department of Defence, in reply to a communication of September 15 last, advising that the rate of payment to officers of the Australian Army Medical Corps for conducting the medical examination of non-flying personnel applying for admission to the Royal Australian Air Force had been reconsidered and that it had been approved that in future a fee of 10s. 6d. per examination would be paid. Previously the fee was 5s. and this had appeared to the Federal Committee to be inadequate.

Hand-book for Recently Qualified Medical Practitioners.

At the request of the Council of the New South Wales Branch the question was considered of publishing a hand-book supplying information to recently qualified medical practitioners in Australia similar to that published in May, 1923, by the British Medical Association for recently qualified practitioners in the United Kingdom. It was recognized that such a hand-book would be of great use to new graduates in Australia and the matter was referred to the Directors of the Australasian Medical Publishing Company, Limited, for their consideration.

Homœopathy.

In an enunciation of principles of ethics in medical practice, confirmed by the Federal Committee in February, 1914, it was declared to be unethical for a practitioner to designate his practice as based on an exclusive dogma such as that of homœopathy. The Committee was asked by the Council of the Victorian Branch to give further consideration to this pronouncement. The ethical position of a practitioner professing to conduct his practice in accordance with the doctrine of homœopathy was discussed and it was decided not to alter the pronouncement.

Medical Insurance Agency.

At the instance of the South Australian Branch Council, the question was considered of forming a medical insurance agency, with agencies in the several States to provide insurance of all kinds including medical defence. The question was discussed in connexion with another proposal by the South Australian Branch for establishing a Federal medical defence union. It was noted that there was a medical defence union in each of the States. The position of medical practitioners in relation to insurance companies in respect of risks specially appertaining in their practices and their homes was discussed and the constitution and objects of the "Medical Insurance Agency" which was formed in England by *The British Medical Journal* and *The Lancet* acting together in 1907, were referred to. By the description of this Agency given in the "British Medical Association Annual Hand-book," 1923-4, pages 222-4, it was seen that its work covered life insurance, savings fund and educational policies for the young, sickness and accident policies and household policies (fire, burglary, workmen's compensation and special perils), as well as motor car insurance and that it had been very successful in the interests of those insured through it.

On the motion of Dr. H. S. Newland, seconded by Dr. W. N. Robertson, the matter was referred to the Branches for consideration and directions were given for inquiries to be made.

Cooperation of the Medical Profession with the Commonwealth Department of Health.

The Committee had had before it at its previous meeting a report in reference to the question of bringing the general practitioner into more active cooperation with the health authorities in preventive measures. Suggestions for the amendment of the report had been made and it was submitted again for further consideration.

Lack of space prevents publication of the report in this issue. It is intended to publish it next week.

Dr. Newland in presenting the report advised that it should be laid before all the Branches so that it might be discussed and the necessity for extended and properly coordinated measures of preventive medicine understood and appreciated. In any remodelling of administration care would have to be taken to avoid centralization. The difficulty in doing what was required arose from the fact that State Governments could not deal with all the questions involved nor could the Commonwealth Government. The most radical change proposed was, he thought, the abolition of the part-time medical officers of health. After discussion a few verbal amendments were made and it was resolved, on the motion of Dr. R. H. Fetherston, seconded by Dr. W. N. Robertson:

> (i.) That the report be forwarded to the Branches for consideration and that suggestions made by the Branches be considered by the Federal Committee at its next meeting.
>
> (ii.) That the Branches be invited to make suggestions for the improvement of the present State *Acts* governing public health.

Royal Commission on National Insurance.

A letter addressed to Dr. Alfred Cox, written by direction of the previous meeting, was read in reference to his articles published in *The Journal of the American Medical Association* of May 7, 14 and 21, 1921, entitled "Seven Years of National Health Insurance in England" and inviting him to write a series of articles on the same subject for the information and guidance of practitioners in the Commonwealth. It was suggested that a suitable medium for their publication would be THE MEDICAL JOURNAL OF AUSTRALIA.

A short report from the Sub-Committee appointed at the previous meeting to collect information on national health insurance was read and received. It was noted that evidence from members of the Association in New South Wales and Queensland had been taken by the Commission and that it was proposed shortly to take evidence from representatives of the Victorian Branch, after which, it was understood, the Commission's report, so far as it concerned compulsory individual medical attendance, would be proceeded with. It was anticipated that the Commission would recommend to the Federal Parliament some scheme of medical benefit.

At the invitation of the Committee, Dr. Newman Morris who had been associated with the Sub-Committee, attended the meeting and gave information in regard to the intentions of the Commission with which the Secretary of the Commission had supplied him. It was made clear that the Report of the Commission would be made at an early date and not, as was previously understood, after eighteen months or two years; so that, if steps were to be taken to ascertain the views of the profession generally throughout the Commonwealth, it would be necessary to act promptly. Reference was made to the report, published in the Supplements to *The British Medical Journal* of January 5, 12 and 19, 1924, of the proceedings before the Court appointed to inquire and report to His Majesty's Government what should be the amount of the capitation fee (per insured person *per annum*), to be calculated as from January 1, 1924, under the *National Insurance Act* in the United Kingdom and it was arranged that the Supplements containing the report should be sent to the Royal Commission for its information.

The Chairman drew the attention of the Committee to a *questionnaire* designed by the Council of the Victorian Branch for submission to the individual members of the Branch and to a proposal for the contribution to THE MEDICAL JOURNAL OF AUSTRALIA of some articles on the subject of compulsory medical benefit which, it was thought, would interest members and assist them in formulating their individual views. Attention was also drawn by the Chairman to an authoritative article, published in *The British Medical Journal* of June 20, 1908, on German hospitals, which set out also the provisions made for domiciliary medical attendance under the national scheme in operation in Germany before the war. Reference was made to the previous work of the Federal Committee in 1914 in supplying answers to the questions submitted by the Commonwealth Statistician in Ooctober, 1913, in connexion with the proposal to introduce a scheme of social insurance into Australia; also to the work subsequently done by the Committee in regard to the formulation of a scheme for a national medical service. In reference to the question of the necessity for the Government to provide a national scheme for medical attendance, comparable to that established in the United Kingdom under the *National Insurance Act* in force there, it was mentioned that the Government had been advised, rightly or wrongly, that there were some half-million people in the Commonwealth to whom medical attendance at the present time was not available.

After prolonged discussion it was resolved—

(i.) On the motion of Dr. W. N. Robertson, seconded by Dr. H. S. Newland:

> That a letter be addressed to the Royal Commission on National Insurance stating that the Federal Committtee is collecting information on national (health) insurance and asking that the completion of the Commission's report be postponed until after evidence now being collected by the Federal Committee has been placed before it.

(ii.) On the motion of Dr. R. H. Fetherston, seconded by Dr. J. Lockhart Gibson:

> That a letter be sent to the Prime Minister (Mr. Bruce) reminding him of a promise of Mr. (now Sir) Joseph Cook in 1913, then Prime Minister, not to introduce any legislation dealing with matters in which the medical profession in its relations with the public is concerned, until the Federal Committee has had time to consider the proposals of the Government and to make suggestions in regard to them.

(iii.) On the motion of Dr. J. Lockhart Gibson, seconded by Dr. W. N. Robertson:

> That Dr. Newman Morris be asked if he will make a résumé of the medical evidence already taken in Australia by the Royal Commission on National Insurance and arrange for its publication in THE MEDICAL JOURNAL OF AUSTRALIA.

(iv.) On the motion of Dr. W. N. Robertson, seconded by Dr. J. A. Dick:

> That the several Branches be asked to send out to their members a *questionnaire* similar to that of the Victorian Branch with a suitable preamble.

Resignation of Dr. F. S. Hone.

In welcoming Dr. Bronte Smeaton to the Committee as a new member representing the South Australian Branch, reference was made to the resignation of Dr. F. S. Hone who, after he had been elected as a representative of the South Australian Branch for the year 1924, had found it necessary to resign. On the motion of Dr. W. N. Robertson, seconded by Dr. J. A. Dick, the following resolution in appreciation of Dr. Hone's services to the Committee was passed:

> That the Federal Committee desires to place on record its great appreciation of the valuable services rendered to the Committee since its inception by Dr. Hone. His wide outlook and statesmanlike grasp of the problems before the Committee have been of inestimable value. The Committee deeply regrets that he has been compelled to relinquish his membership because of serious illness in his

family and it hopes that his anxiety may be speedily relieved and that at no distant date he may be able to resume his activities on the Federal Committee.

Sir Lindo Ferguson.

It was resolved on the motion of Mr. G. A. Syme, seconded by Dr. R. H. Fetherston:

That a letter be sent to Sir Henry Lindo Ferguson, Dean of the Faculty of Medicine in the University of Otago (New Zealand), congratulating him on behalf of the Federal Committee of the British Medical Association in Australia, upon the honour of knighthood recently conferred upon him.

Votes of Thanks.

A vote of thanks was passed to the Council of the Victorian Branch for their kindness in providing accommodation for the meeting of the Committee at the Medical Society Hall and for their courtesy in entertaining the members of the Committee at dinner at Windsor Hotel on February 25.

A vote of thanks was also passed to the Chairman, Mr. G. A. Syme, for presiding at the meeting and for his kindness in entertaining the members of the Committee at luncheon on February 26.

Next Meeting of the Federal Committee.

It was decided that the next meeting of the Federal Committee should be held in Sydney late in July, the exact date to be determined by the Chairman.

NOMINATIONS AND ELECTIONS.

THE undermentioned has been nominated for election as a member of the New South Wales Branch of the British Medical Association:

CALLEN, AUSTIN ARTHUR, M.B., Ch.M., 1922 (Univ. Sydney), Nimbin.

Post-Graduate Work.

SPECIAL POST-GRADUATE LECTURES IN MELBOURNE.

THE HONORARY SECRETARIES OF THE MELBOURNE PERMANENT COMMITTEE FOR POST-GRADUATE WORK announce that it is the intention of the Committee to arrange for a course of post-graduate lectures to be delivered in August or September of this year on "Some Disorders of Renal Function." The fee for the course will be two guineas. Fuller details of these lectures will be published at a later date.

Correspondence.

FEDERAL INCOME TAX DEDUCTIONS.

SIR: *Re* Federal income tax deductions and your comments in the journal of February 9, 1924.

My experience does not tally with your remarks or the article by Messrs. H. L. Cunningham and R. J. Stiffe, in regard to depreciation allowed on motor cars. I will mention it and would advise all medical men to put in their claims for deductions as I did, as it appears probable that they will be allowed. In January, 1922, I was using a Ford car which on July 1, 1921, showed a Federal tax valuation of £155. On March 10, 1922, I purchased a Fiat for for £540 and a few days later I sold the Ford for £140 net. In my income tax return, 1921-1922, I claimed £15 depreciation on the Ford, as being difference in estimated value on July 1, 1921 and the price I received for the car. I also claimed £16 depreciation on the Fiat car for the period March 10 to June 30, 1922, or a total of £31. I was allowed: Ford, £155, 10%, eight months, £10; Fiat, £540, 10%, four months, £18; total, £28—*id est* £3 less than I claimed, but giving full 10% allowance on the Fiat which was purchased during the year for which the return was made and which from a common sense point of view is only rational and right, as a motor car depreciates as soon as it is put in use and relatively a great deal more in the first few months when it passes from "new" to "second-hand," than later on.

Yours, etc.,
F. J. V. BONNIN, M.D.

Talora, Ararat, Victoria,
February 11, 1924.

PUBIOTOMY.

SIR: I have been particularly interested in Dr. O'Brien's article on "Pubiotomy" in your issue of February 23, 1924. Would Dr. O'Brien give your readers his opinion on the value of induction of labour, say, two to four weeks early, as opposed to pubiotomy; also the functional after-results in the mothers as regards locomotion?

Yours, etc.,
E. BRETTINGHAM-MOORE.

149, Macquarie Street, Hobart, Tasmania,
February 26, 1924.

HERPES ZOSTER AND VARICELLA.

SIR: Dr. le Feuvre's contempt (*The British Medical Journal*, January 5, 1924, page 1277) for "generalized *herpes zoster*" cannot fail to interest me. I have met with one case only that I thought came under that head. This was a case of a man of thirty-five. He had a typical *herpes zoster* along the course of the left sixth nerve. Besides that a crop of vesicles was scattered all over the trunk including the right side and particularly on the face. But all along the track of the sixth nerve it was most marked. This case occurred twelve years ago. Though the man lived in a house full of children, none of them had varicella afterwards, nor was there any epidemic present at the time. Quoting Dr. le Feuvre, he says: "A course of arsenic may induce an attack of shingles and the latter may be followed by chicken-pox." This strikes me as rather a bold statement, especially when he goes on to say that we thus may have "an artificially induced epidemic of an infectious disease," *id est* a local complaint in one can start a germ disease in others. This seems to me to be impossible.

Dr. Pollitzer, one of the leading dermatologists of New York, reports several cases of *herpes zoster generalisatus* then occurred in a small-pox scare. Dr. Herman Lawrence, of Melbourne, has just told me of a case he saw yesterday. She had *herpes zoster*, but four years ago she suffered from an attack of varicella. I am afraid that *herpes zoster generalisatus* is not dead after all.

Yours, etc,.
G. HORNE.

63, Collins Street, Melbourne,
Undated.

MALARIA TREATMENT OF SYPHILIS OF THE NERVOUS SYSTEM.

SIR: On account of the adoption of the Gerstmann method of this form of treatment on the Continent and more recently in England (*vide The British Medical Journal*) a description of the method adopted by Gerstmann himself may be of value. The treatment is the result of experience in the nerve clinic of Wagner-Jauregg in Vienna and is really the evolution of methods used for the past twenty years. At first he experimented with old tuberculin with the object of increasing the temperature,

but found later that it acted best in conjunction with mercury and "Arseno-benzene" ("Salvarsan") preparations. Later typhoid vaccine was used and by an accidental discovery in his clinic he found that a patient after an attack of tertian malaria was much improved of his long-standing peneral paresis.

Gerstmann, an assistant in the clinic, quotes seven hundred cases of neuro-syphilis since 1917, with extraordinarily good results and those cases that have improved at first, seldom relapse. As a matter of fact a great proportion are now back to work and only a small proportion are not benefited.

It has been remarked that paretics seldom had an infectious disease previously and the therapeutic inoculation of tertian malaria into a neuro-syphilitic is an imitation of Nature's way of making the cerebro-spinal tissues more resistant to the *spironema pallidum*. It may be here mentioned that the rise in temperature incident on an attack of tertian malaria is not the only factor in the improvement of the condition.

In this clinic less reliance is placed on the old classification of syphilis of the brain into secondary and tertiary lesions as to: (i.) Interstitial lesions, which include those of the meninges and blood vessels and (ii.) parenchymatous.

In the former case arsenic and mercury are given in addition to the malaria, but it is pointed out that they are of little value in the latter on account of the power of the chorioid plexus to filter from the blood stream large metallic molecules, so that these powerful drugs are unable to be brought into contact with spironemata which lie in contact with nerve cells bathed by the cerebro-spinal fluid.

Intravenous therapy of "Arseno-benzene" in neuro-syphilis had been proved to be of little value and has given place to various other methods which also were discarded by Gerstmann. The more important of these were: (*a*) Injection of Salvarsanized serum into the thecal cavity—method of Swift Ellis, of America; (*b*) injection of small and frequent doses of "Salvarsan" into the thecal cavity—method of Gennerich, of Germany. The objection to both methods is that substances introduced intrathecally are rapidly absorbed into the blood stream.

It was found that the results of intra-spinal therapy did not differ from mercury intra-muscularly or "Arseno-benzene" intravenously. Partial Wassermann reactions are considered as negatives. When the lesions are regarded as meningo-vascular, mercury and "Arseno-benzene" are given, the mode of therapy being as follows: "Neo-Salvarsan" is administered for the first two doses 0.3 gramme and if it is borne well, it is increased to 0.45 gramme and then to 0.6 gramme—ten doses in all. Mercury is given as succinamide at two daily intervals for thirty doses. Thus mercury succinamide on Monday, Wednesday and Friday and "Neo-Salvarsan" on Saturday.

Women are not given more than 0.45 gramme of "Arseno-benzene."

The method of malaria inoculation is as follows:

A patient suffering from tertian malaria is selected. He must not have been treated previously with quinine. One cubic centimetre of blood is extracted from the median basilic or other suitable vein and is straightaway injected into the subcutaneous tissues of the interscapular tissues of the paretic. The needle is pushed in various directions while in the subcutaneous tissues in order to give a larger absorbing area. After an incubation period of one to two weeks the patient has attacks of tertian fever. He is allowed to have eight to twelve of these attacks and is then given quinine *per os* in doses of 0.5 gramme quinine bisulphate twice daily for three days and then once a day for six days. The higher the fever, the better the treatment. At first the blood was taken from the donor during an attack, but it was found later that it did not matter.

Professor Kyrle in the Finger Clinic here quotes four hundred cases with best results and uses this treatment when the cerebro-spinal fluid shows a positive Wassermann, even in the secondary stagee of syphilis, where there are no symptoms of cerebro-spinal damage. He goes so far as to say that this is the ideal stage for the malaria treatment. He says that there have been no catastrophes. He injects five cubic centimetres of the donor's blood subcutaneously or intravenously and allows eight to ten attacks, and stops the action of the malaria by 0.5 gramme quinine hydrochloride twice daily for ten days or 5 cubic centimetres of a 10% quinine bisulphate solution intravenously and repeats the dose in eight hours. An injection of 0.45 gramme of "Neo-Salvarsan" is administered next day and on the third day the two doses of quinine bisulphate are repeated.

During the treatment with malaria the patients lose weight up to twelve pounds, but after a few doses of 0.45 gramme "Neo-Salvarsan" the patient "blooms again."

Both observers note that the cerebro-spinal fluid does not show an alteration for some weeks and improvement can be noted in paretic cases for months after.

Those cases that have a positive cerebro-spinal fluid for years, notwithstanding copious administrations of "Arseno-benzene" preparations, bismuth and mercury, have been markedly affected with the malaria treatment.

Both Gerstmann and Kyrle declare that improvement has been shown in 70% of cases of advanced nervous disease, such as general paresis, tabo-paralysis and in infantile conditions, though the earlier the stage the better the result. Remissions from symptoms are of two types: (i.) Complete where the mental and motor symptoms disappear and the patient goes back to work. In this class he appears to be cured. It is seldom, however, that the Argyll-Robertson pupil disappears and the knee-jerks return. (ii.) Incomplete where there is improvement, but there are residual symptoms which depend on the stage of the disease and the destruction of nerve structures. It must, of course, be noted that the tertian and not the tropical form of malaria must be used.

Though both the observers say that no danger has resulted from the malaria treatment and everyone else in Europe who has tried the method, speaks so highly of the method, Professor J. Erdheim, pathologist to the *Allegemeine Krankenhaus*, of this city, states that he has seen some deaths, fortunately very few, as a result, not of the malaria itself, but from the combination of fever *plus* the degenerated nervous structures.

Yours, etc.,

D. ROSENBERG, M.B., B.S .(Melbourne).

Vienna, Austria,
February 1, 1924.

AN OUTBREAK OF TYPHOID FEVER.

SIR: I am forwarding the following notes on a small outbreak of typhoid fever, in the hope that they may be worth publishing as of interest to medical officers of health.

Colac is a town of about four thousand five hundred inhabitants, situated at the southern end of Lake Colac, in the shire of the same name, in the south-western district of Victoria. The surrounding dairying and agricultural country is fairly closely settled. The town itself is well sited on undulating ground drained by the Barongarook Creek which flows north through the town into the lake. The water supply is abundant and good, being derived from the Olangolah River near Mount Sabine, the catchment being in uninhabted forest country. There is a system of municipal garbage removal from the main portion of the town. The town is as yet unsewered (but a sewerage authority has been constituted and has taken the initial steps towards sewering the town). Night soil is removed by contract. The double pan system is well carried out, but closets generally, as is usual with country towns, are not fly-proof.

Typhoid fever is not common. There were no cases in 1920, eight in 1921, three in 1922, none in 1923 till November, when the following five cases occurred, within a few weeks of each other:

A.X., *ætatis* two years and two months, became ill on November 23, 1923, at first diagnosed as pneumonia.

B.X., *ætatis* four years, brother of A.X., became ill on December 3, 1923; definite diagnosis of both as typhoid fever on December 8, 1923.

C.Y., *ætatis* eight years, became ill on November 17, 1923, definite diagnosis made on December 8, 1923.

D.Z., adult, became ill on December 18, 1923, definite diagnosis on December 25, 1923.

E.Z., *ætatis* sixty years, husband of D.Z., became ill on January 2, 1924, and was diagnosed on January 9, 1924.

The X. household occupies a comfortable wooden villa on the south-east edge of the town. The family consists of Mr. X., who had typhoid fever fifteen years ago, Mrs. X. (no history of typhoid), F.X., *ætatis* ten years, who had typhoid six years ago and the two patients.

Sanitary conditions good, except that the closet is not fly-proof. A trained nurse was in charge of the cases which were nursed at home.

The Y. household consists of the patient and his parents who gave no typhoid history. They occupy a good class dwelling about half a mile from the X. household, and nearer the middle of the town; sanitary conditions were good.

The Z. household, in the same street as the X. household, consists of the two patients and one daughter; they have been in Colac about two years. No typhoid history.

The common factor in all three households was the milk supply. Both the X. and Z. households normally obtain their milk from Mrs. H., a neighbour, who kept two cows and sold milk to the people around. The Y. household usually kept their own cow, but from October to December had obtained milk from Mrs. H.

Suspicion was directed to the milk supply both on this account and because it was found on inquiry that in 1921 several of the cases of typhoid which occurred then were in this neighbourhood and were customers of Mrs. H. Of these, a Mrs. K. and her son, *ætatis* seven years, were still in the neighbourhood.

The H. household consists of Mr. H., who gave a history of having had typhoid seventeen years before; Mrs. H., a son and a daughter. (No typhoid history from these three.)

On December 12, 1923, blood specimens were taken from Mr. X., Mr. H., Mrs. H. and their son, F.X., Mrs. K. The first three gave a strong partial Widal, the last three were negative. On the same date urinary specimens from Mr. X. and Mr. H. were sent. Both of these were negative.

Further blood specimens from Mr. X., Mr. and Mrs. H. resulted in Mr. X. and Mr. H. being reported as positive Widals; Mrs. H. negative.

Urinary specimens from Mr. and Mrs. H. were both negative. Specimens of stools of both Mr. and Mrs. H. were sent and those of Mr. H. were found to contain *Bacillus typhosus*.

There is no evidence that Mr. H. took any part in the handling of the milk. The premises generally were kept clean, but there was no proper separator room, no effort to keep flies from having access to the milk. The separator was housed in the bathroom. The closet, about fifty yards from the house, was dilapidated and not fly-proof. Flies were fairly plentiful. It should be mentioned that this place was not registered as a dairy.

The following action has been taken. An order has been issued to Mrs. H. under the Infectious Diseases Regulations, by the Municipal Officer of Health, forbidding the sale of milk or milk products from the premises. They have been instructed to render the house and closet fly-proof, the sanitary contractor has been instructed to treat the pan contents as though from a house where there is a case of typhoid, *id est* by incineration. The carrier, Mr. H., has been instructed to place himself under his usual medical attendant for treatment.

So far no further cases have occurred.

I am indebted to Mr. A. Brown, Municipal Officer of Health of the shire of Colac, and Dr. K. Doig, who attended the cases and took most of the specimens, for the above information. Widals *et cetera* were carried out by the University Laboratory.

Yours, etc.,

GEORGE COLE, D.S.O., M.B., B.S., D.P.H.
District Health Officer, Western District.

Warrnambool,
March 7, 1924.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

A MEETING of the Senate of the University of Sydney was held on March 3, 1924.

The following appointments were made for the year 1924-25: Dr. V. M. Coppleson, part-time demonstrator in anatomy; Dr. V. M. Coppleson and Dr. W. N. Horsfall, part-time demonstrators in physiology; Mr. C. J. M. Walters, B.V.Sc., M.B., Ch.M., temporary lecturer in veterinary *materia medica* and therapeutics; Mr. I. Clunies Ross, B.V.Sc., temporary lecturer in veterinary parasitology. Mr. J. Kenner, D.Sc., Ph.D., was appointed to the Chair of Organic Chemistry, Pure and Applied.

Obituary.

GEORGE ROBERT ADCOCK.

IT is with regret we have to announce the death of Dr. G. R. Adcock which occurred suddenly at Evandale, Tasmania, on February 27, 1924.

ARTHYR JOHN NYULASY.

WE regret to announce the death of Dr. A. J. Nyulasy, of Perth, which occurred at the residence of his brother, Dr. Frank Nyulasy, Toorak, Melbourne, on February 28, 1924..

WALTER ELI HARRIS.

THE announcement of the death of Dr. Walter Eli Harris which occurred at Armidale on March 10, 1924, is made with regret.

Naval and Military.

APPOINTMENTS.

THE following appointments, changes, *et cetera* have been promulgated in *Commonwealth of Australia Gazette*, No. 16 of March 6, 1924:

Australian Military Forces.

FIRST MILITARY DISTRICT.

Australian Army Medical Corps.

To be Lieutenant (provisionally)—PETER NEWTON MACGREGOR, 1st February, 1924.

SECOND MILITARY DISTRICT.

CAPTAIN AND BREVET MAJOR G. C .WILLCOCKS, O.B.E., M.C., is transferred to the Reserve of Officers and to be Major, 1st February, 1924.

Australian Army Medical Corps Reserve.

HONORARY CAPTAIN W. SHORTT is placed on the Retired List, with the rank of Captain and with permission to wear the prescribed uniform, 18th February, 1924.

THIRD MILITARY DISTRICT.

Australian Army Medical Corps.

To be Captain (provisionally)—DAVID LESLIE YOFFA, 1st January, 1924. The provisional appointment of CAPTAIN D. L. YOFFA is terminated under the provisions of Section 15 of the Defence Act, 31st December, 1923.

FOURTH MILITARY DISTRICT.

Australian Army Medical Corps Reserve.

HONORARY CAPTAIN R. H. N. CONNELL is transferred from the Australian Army Medical Corps Reserve, 5th Military District, 1st March, 1924.

Award of the Colonial Auxiliary Forces Long Service Medal.

Australian Army Medical Corps.

COLONEL S. R. BURSTON, C.B.E., D.S.O.

FIFTH MILITARY DISTRICT.

Australian Army Medical Corps Reserve.

HONORARY CAPTAIN R. H. N. CONNELL is transferred to the Australian Army Medical Corps Reserve, 4th Military District, 1st March, 1924.

Corrigendum.

PROFESSOR W. A. OSBORNE has called our attention to a typographical error in our report of the Mathison Lecture published in the issue of March 1, 1924. In the last line on page 211, the word "roving" should be "saving," so that the sentence should read: "This 'heart saving' action is undoubted, but I am never able properly to interpret the meeting of the phenomenon."

Books Received.

HERNIA: ITS ANATOMY, ETIOLOGY, SYMPTOMS, DIAGNOSIS, DIFFERENTIAL DIAGNOSIS, PROGNOSIS, AND OPERATIVE TREATMENT, by Leigh F. Watson, M.D.; 1924. St. Louis: C. V. Mosby Company; Royal 8vo., pp. 660, with two hundred and thirty-two original illustrations by W. C. Shepard. Price: $11.00.

Medical Appointments.

DR. C. PURSER (B.M.A.) has been appointed Chairman of the Board of Directors of the Royal Prince Alfred Hospital, Camperdown, Sydney.

* * * *

DR. RUBY TOWNSEND (B.M.A.) has been appointed member of the Council of the Box Hill Technical School (Victoria), for the period ending December 31, 1924.

* * * *

DR. PAUL ERNEST VOSS, M.C. (B.M.A.) has been appointed Government Medical Officer at Rockhampton and Health Officer for the purpose of the *Health Acts*, 1900 to 1922, during the absence of Dr. Francis Henry Vivian Voss (B.M.A.).

* * * *

DR. ANTON BREINL (B.M.A.) has been appointed Trustee of the Grammar School, at Townsville, Queensland.

* * * *

DR. H. B. ELLERTON, DR. G. P. DIXON, C.B.E. (B.M.A.), and DR. C. M. LILLEY (B.M.A.) have been appointed members of the Nurses' Registration Board, Queensland, for a period of three years.

* * * *

DR. HERBERT CHAMPION HOSKING has been appointed Junior Medical Officer, Parkside Mental Hospital, South Australia.

* * * *

DR. C. E. DORSCH has been appointed Officer of Health for the district of Morgan, South Australia, *vice* Dr. A. H. Guymer (B.M.A.), resigned.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429, Strand, London, W.C.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney	Australian Natives' Association Ashfield and District Friendly Societies' Dispensary Balmain United Friendly Society's Dispensary Friendly Society Lodges at Casino Leichhardt and Petersham Dispensary Manchester Unity Oddfellows' Medical Institute, Elizabeth Street, Sydney Marrickville United Friendly Societies' Dispensary North Sydney United Friendly Societies People's Prudential Benefit Society Phœnix Mutual Provident Society
VICTORIA: Honorary Secretary, Medical Society Hall, East Melbourne	All Institutes or Medical Dispensaries Australian Prudential Association Proprietary, Limited Mutual National Provident Club National Provident Association
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane	Brisbane United Friendly Society Institute Stannary Hills Hospital
SOUTH AUSTRALIA: Honorary Secretary, 12, North Terrace, Adelaide	Contract Practice Appointments at Renmark Contract Practice Appointments in South Australia
WESTERN AUSTRALIA: Honorary Secretary, Saint George's Terrace, Perth	All Contract Practice Appointments in Western Australia
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington	Friendly Society Lodges, Wellington, New Zealand

Diary for the Month.

MAR. 18.—New South Wales Branch, B.M.A.: Medical Politics Committee; Organization and Science Committee.
MAR. 19.—Victorian Branch, B.M.A.: Council.
MAR. 19.—Western Australian Branch, B.M.A.: Branch.
MAR. 19.—South Sydney Medical Association, New South Wales.
MAR. 25.—New South Wales Branch, B.M.A.: Council (Quarterly).
MAR 27.—New South Wales Branch, B.M.A.: Annual Meeting.
MAR. 28.—Queensland Branch, B.M.A.: Council.
APR. 2.—Victorian Branch, B.M.A.: Branch.
APR. 4.—Queensland Branch, B.M.A.: Branch.
APR. 9.—Tasmanian Branch, B.M.A.: Branch.
APR. 9.—Melbourne Pædiatric Society.
APR. 10.—Brisbane Hospital for Sick Children: Clinical Meeting.
APR. 11.—Queensland Branch, B.M.A.: Council.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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